ENTRY D64718 #type complete **iProClass View of D64718**
TITLE conserved hypothetical protein HP1588 - Helicobacter pylori (strain 26695)
ORGANISM #formal_name Helicobacter pylori
#cross-references taxon:210
DATE 09-Aug-1997 #sequence_revision 09-Aug-1997 #text_change 08-Oct-1999
ACCESSIONS D64718
REFERENCE A64520
#authors Tomb, J.F.; White, O.; Kerlavage, A.R.; Clayton, R.A.; Sutton, G.G.; Fleischmann, R.D.; Ketchum, K.A.; Klenk, H.P.; Gill, S.; Dougherty, B.A.; Nelson, K.; Quackenbush, J.; Zhou, L.; Kirkness, E.F.; Peterson, S.; Loftus, B.; Richardson, D.; Dodson, R.; Khalak, H.G.; Glodek, A.; McKenney, K.; Fitzegerald, L.M.; Lee, N.; Adams, M.D.; Hickey, E.K.; Berg, D.E.; Gocayne, J.D.; Utterback, T.R.; Peterson, J.D.; Kelley, J.M.; Cotton, M.D.; Weidman, J.M.; Fujii, C.; Bowman, C.; Watthey, L.; Wallin, E.; Hayes, W.S.; Borodovsky, M.; Karpk, P.D.; Smith, H.O.; Fraser, C.M.; Venter, J.C.
#journal Nature (1997) 388:539-547
#title The complete genome sequence of the gastric pathogen Helicobacter pylori.
#cross-references MUID:97394467; PMID:9252185
#accession D64718
##status preliminary; nucleic acid sequence not shown; translation not shown
##molecule_type DNA
##residues 1-253 ##label TOM
##cross-references GB:AE000656; GB:AE000511; NID:g2314771; PIDN:AAD08627.1; PID:g2314773; TIGR:HP1588
CLASSIFICATION SF028794
SUMMARY #length 253 #molecular_weight 28417

SEQUENCE

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   61 I A E E L Q Y Y G S N S F A S F I K G E G V L Y K E I L C D
   91 V C D K L K V N Y N K K T E T T L I E Q N M L S K I L E R S
  121 L E E M D D E E V K E M C D E L S I K N T D N L N R Q A L S
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  181 G R G L S L A G N Q V L T R T L S F L T G P V G W I I T G V
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[redacted] section of the Protein Sequence Database, release 79.02, 09-May-2004, assembled and annotated by the PIR-International.

ALIGNMENTS containing D64718:
M40954 040954 family - 0.0 0.0

WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Wednesday, July 07, 2004

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☐	L4	L3 and (sds or sdspage or electrophoresis)	14
☐	L5	pylori.clm.	352
☐	L6	L5 and (rmw or mw or kd or kda or k-da or dalton or kilodalton or kilo-dalton or da or size).clm.	28
☐	L7	calenoff.in. and pyloir	0
☐	L8	calenoff.in. and pylori	1

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 28 of 28 returned.

1. 6709656. 10 Feb 99; 23 Mar 04. Helicobacter pylori adhesin binding group antigen. Boren; Thomas, et al. 424/190.1; 424/184.1 424/234.1 435/252.1 435/7.32 514/25 530/350. A61K039/02.

2. 6699722. 13 Apr 01; 02 Mar 04. Positive detection lateral-flow apparatus and method for small and large analytes. Bauer; Jeffrey S., et al. 436/518; 422/56 422/57 435/7.1 435/7.92 436/514 436/541. G01N033/543.

3. 6616938. 06 Jun 01; 09 Sep 03. Method of making chewing gum containing colloidal bismuth subcitrate. Athanikar; Narayan K.. 424/440; 424/653. A61K009/68 A61K033/24.

4. 6607742. 13 Mar 02; 19 Aug 03. Suppository composition which is a spray-dried suspension of an acid-labile proton pump inhibitor in a solvent solution of a sterol and polymer. Linder; Rudolf, et al. 424/436; 424/434 514/338. A61F013/00 A61F002/00.

5. 6517809. 01 Aug 00; 11 Feb 03. Process for preparing a reactive pharmaceutical product for the detection of gastrointestinal disorder caused by bacteria in the gastrointestinal superior tract. Marshall; Barry J.. 424/1.37; 424/1.25 424/1.29 424/1.33 424/1.81. A61K051/00.

6. 6468739. 11 Mar 98; 22 Oct 02. Process for identifying secretory genes from helicobacter pylori. Haas; Rainer, et al. 435/6; 435/320.1 435/69.1 435/69.8 536/23.1 536/23.7. C07H021/02 C07K001/00 A61K039/02.

7. 6468534. 21 Sep 00; 22 Oct 02. Methods for obtaining transfer factor from avian sources, compositions including avian-generated transfer factor, and methods of use. Hennen; William J., et al. 424/157.1; 424/130.1 424/184.1 424/201.1 424/204.1 424/227.1 435/41 530/300 530/350. A61K039/395 A61K039/00 A61K039/12 C12P001/00 C07K001/00.

8. 6464625. 23 Jun 99; 15 Oct 02. Therapeutic method and apparatus for debilitating or killing microorganisms within the body. Ganz; Robert A.. 600/3;. A61N005/00.

9. 6403099. 01 Nov 94; 11 Jun 02. Conjugates formed from heat shock proteins and oligo-or polysaccharides. Rappuoli; Rino, et al. 424/248.1; 424/192.1 435/12 435/7.32 514/569 530/395. C01B003/00 G01N033/554 C07K001/00.

10. 6399629. 01 Jul 98; 04 Jun 02. Efflux pump inhibitors. Chamberland; Suzanne, et al. 514/313; 514/408 514/423 546/159 548/518 548/537. A61K031/47 A61K031/40 C07D217/18 C07D207/00.

11. 6319513. 27 Jul 99; 20 Nov 01. Oral liquid mucoadhesive compounds. Dobrozsi; Douglas Joseph. 424/434; 424/435. A61F013/02.

12. 6113875. 29 May 97; 05 Sep 00. Diagnostic preparation for detection of helicobacter pylori. Nystrom; Christer, et al. 424/1.29; 424/1.25 424/1.33 424/1.81 424/9.1. A61K051/00 A61K049/00.

13. 6086893. 05 Dec 97; 11 Jul 00. Helicobacter lactoferrin receptor. Dupuy; Monique, et al. 424/234.1; 435/252.1 435/6 435/7.32. A61K039/02.

☐ 14. 6068985. 26 Apr 95; 30 May 00. In vitro test for Helicobacter pylori. Cripps; Allan, et al. 435/7.32; 424/234.1. G01N033/554.

☐ 15. 6054134. 26 Jul 96; 25 Apr 00. Haemophilus adhesin protein. Lingwood; Clifford A.. 424/256.1; 424/185.1 424/190.1 424/200.1 424/234.1 424/241.1 435/69.1 435/69.3 530/350 536/23.1 536/23.7. A61K039/102.

☐ 16. 5972389. 19 Sep 96; 26 Oct 99. Gastric-retentive, oral drug dosage forms for the controlled-release of sparingly soluble drugs and insoluble matter. Shell; John W., et al. 424/501; 424/426. A61K009/16 A61K047/34.

☐ 17. 5866375. 06 Jun 95; 02 Feb 99. Method for the culture of microorganisms of the genera helicobacter, campylobacter and arcobacter employing culture media comprising cyclodextrins. Figura; Natale, et al. 435/71.3; 435/71.1 435/71.2 435/822 530/825. C12P021/00.

☐ 18. 5846751. 07 Jun 95; 08 Dec 98. Test kits and methods for detecting H. pylori. Pronovost; Allan David, et al. 435/7.32; 435/7.2 435/7.92 435/7.93. G01N033/554 G01N033/53.

☐ 19. 5814455. 18 Aug 94; 29 Sep 98. Antigen preparation for detecting H. pylori. Pronovost; Allan David, et al. 435/7.1; 424/184.1 424/234.1 435/7.2 435/7.92 435/822 514/2. G01N033/53 G01N033/536 G01N033/569 C12N001/00.

☐ 20. 5733740. 30 Sep 94; 31 Mar 98. Taga gene and methods for detecting predisposition to peptic ulceration and gastric carcinoma. Cover; Timothy L., et al. 435/7.32; 435/320.1. G01N033/554 G01N033/569.

☐ 21. 5719052. 11 Jan 96; 17 Feb 98. Simple examination method of infection with Helicobacter pylori and device therefor. Ito; Masaharu, et al. 435/287.1; 422/58 422/85 422/86 435/287.7 435/807 600/543 600/562. C12M003/00 G01N021/00 A61B005/08.

☐ 22. 5711936. 05 Jun 95; 27 Jan 98. Ultramulsion based ingestible compositions. Hill; Ira D., et al. 424/49; 424/78.38 514/63. A61K007/16 A61K031/74.

☐ 23. 5582837. 30 May 95; 10 Dec 96. Alkyl-substituted cellulose-based sustained-release oral drug dosage forms. Shell; John W.. 424/451; 424/452 424/457 424/464 424/465 424/468 424/489 514/781 514/786 514/925 514/951. A61K009/16 A61K009/22 A61K009/52.

☐ 24. 5459041. 18 Feb 88; 17 Oct 95. Campylobacter pylori antigens and uses thereof for detection of Campylobacter pylori infection. Blaser; Martin J., et al. 435/7.21; 435/7.3 435/7.92 435/7.93 435/7.94 435/7.95 435/961 435/974 435/975 436/518 436/527 436/528 436/529 436/531 436/533 436/547 436/804 530/350 530/413 530/806 530/810 530/825. G01N033/53.

☐ 25. 5420014. 30 Apr 92; 30 May 95. Rapid in vitro test for helicobacter pylori using saliva. Cripps; Allan, et al. 435/7.32; 435/7.9 435/7.92 435/7.94 435/7.95 436/518 436/527 436/530 436/531. G01N033/569.

☐ 26. 5262156. 12 Aug 91; 16 Nov 93. Antigenic compositions and their use for the detection of Helicobacter pylori. Alemohammad; Mohammad M.. 435/7.32; 435/7.92 435/7.93 435/7.94 435/7.95 436/513 530/350. C07K003/00 C07K013/00 C07K015/00 C07K017/00.

☐ 27. RE34101. 19 Mar 91; 13 Oct 92. Process for preparation of high molecular weight cell-associated protein of Campylobacter pylori and use for serological detection of Campylobacter pylori infection. Evans; Dolores G., et al. 435/7.32; 435/7.1 435/7.92 435/975 530/350 530/806 530/825. C12Q001/00 G01N033/531 G01N033/569.

☐ 28. 4882271. 10 Mar 88; 21 Nov 89. Process for preparation of high molecular weight cell-associated protein of campylobacter pylori and use for serological detection of campylobacter pylori infection. Evans; Dolores G., et al. 435/7.32; 435/12 435/810 436/501 436/518 436/808 436/811 530/350 530/413. G01N033/53 G01N033/543 C12Q001/58 C07K015/00.

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Search Results - Record(s) 1 through 18 of 18 returned.

1. 6747137. 12 Feb 99; 08 Jun 04. Nucleic acid sequences relating to Candida albicans for diagnostics and therapeutics. Weinstock; Keith G., et al. 536/23.1; 435/6 536/24.3 536/24.31 536/24.32 536/24.33. C07H021/00 C12Q001/68.

2. 6737441. 21 Aug 01; 18 May 04. Treatment of helicobacter with isothiocyanates. Fahey; Jed W.. 514/514; 424/755 514/24 514/515. A61K031/27 A61K035/78 A01N047/40.

3. 6734212. 10 Oct 01; 11 May 04. Methods and compositions for treating or preventing bacterial infection. Zhu; Dexu, et al. 514/535; 206/438 514/538 560/34 562/439. A61K031/24 C07C229/00 C07C279/00 A61B017/06.

4. 6709656. 10 Feb 99; 23 Mar 04. Helicobacter pylori adhesin binding group antigen. Boren; Thomas, et al. 424/190.1; 424/184.1 424/234.1 435/252.1 435/7.32 514/25 530/350. A61K039/02.

5. 6689567. 26 May 00; 10 Feb 04. Method for assaying the function of FlaA1 and WbpM. Lam; Joseph S., et al. 435/7.1; 435/232 435/4. G01N035/53 C12Q001/527 C12N009/88.

6. 6670160. 03 May 01; 30 Dec 03. .alpha.1,2-fucosyltransferase. Taylor; Diane, et al. 435/193; 435/183 435/252.3 435/252.33 435/320.1. C12N009/10.

7. 6562958. 04 Jun 99; 13 May 03. Nucleic acid and amino acid sequences relating to Acinetobacter baumannii for diagnostics and therapeutics. Breton; Gary, et al. 536/23.7; 536/23.1. C07H021/02.

8. 6534298. 07 Dec 00; 18 Mar 03. Nucleic acids encoding .alpha.-1,3 fucosyltransferases and expression systems for making and expressing them. Taylor; Diane E., et al. 435/193; 435/252.3 435/254.11 435/254.2 435/320.1 435/325 435/419 536/23.1. C12N009/10 C12N015/54.

9. 6451533. 09 Mar 00; 17 Sep 02. FldA gene and methods for detecting predisposition to mucosa-associated lymphoid tissue lymphoma of the stomach. Wang; Jin-Town, et al. 435/6; 435/5 435/91.2 536/23.1. C12Q001/68.

10. 6410719. 10 Feb 98; 25 Jun 02. Blood group antigen binding protein and corresponding agents. Boren; Thomas, et al. 536/23.7; 536/23.1. C07H021/04.

11. 6399337. 05 Jun 98; 04 Jun 02. .alpha.1,3-fucosyltransferase. Taylor; Diane E., et al. 435/97; 435/72 435/74. C12P019/18.

12. 6322985. 27 Dec 99; 27 Nov 01. Abundant, well distributed and hyperpolymorphic simple sequence repeats in prokaryote genomes and use of same for prokaryote classification and typing. Kashi; Yechezkel, et al. 435/6; 435/91.2 536/23.1 536/24.3. C12Q001/68 C12P019/34 C07H002/104.

13. 6238901. 23 Sep 99; 29 May 01. Type II restriction endonuclease, HPY 188 III, obtainable from Helicobacter pylori J188 and a process for producing the same. Morgan; Richard D., et al. 435/193; 435/199 435/252.3 435/320.1 536/23.2. C12N009/10 C12N009/22 C12N015/54 C12N015/55.

☐ 14. 6238894. 02 Nov 99; 29 May 01. .alpha.1,2 fucosyltransferase. Taylor; Diane, et al. 435/101; 435/193. C12N019/04.

☐ 15. 6133009. 23 Sep 99; 17 Oct 00. Type II Restriction endonuclease, HpyCH4V, obtainable from helicobacter pylori CH4 and a process for producing the same. Morgan; Richard D., et al. 435/199; 435/252.3 435/320.1 536/23.2. C12N009/22 C12N015/55.

☐ 16. 6130069. 24 Mar 99; 10 Oct 00. IspA from Streptococcus pneumoniae. Gwynn; Michael, et al. 435/69.1; 435/252.3 435/320.1 435/325 536/23.1 536/23.2 536/23.4 536/23.5 536/23.7. C12P021/06.

☐ 17. 5260057. 31 Dec 92; 09 Nov 93. Method and product for the treatment of gastric disease. Cordle; Christopher T., et al. 424/157.1; 424/164.1 530/387.1 530/388.4 530/389.1. A61K039/395 C07K015/28 C07K015/00.

☐ 18. 5258178. 07 Aug 92; 02 Nov 93. Method and product for the treatment of gastric disease. Cordle; Christopher T., et al. 424/157.1; 424/164.1 435/170 530/388.4. A61K039/395 C12N015/02 C07K015/28.

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Terms	Documents
26695 and pylori	18

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TABLE 6

COMPARISON OF H. pylori CULTURE REISOLATION AND GRAM STAIN OF MUCUS PIGLETS WITH GRAM STAIN (MUCUS).sup.c FEEDING H. pylori POSITIVE GNR POSITIVE/ PIGLETS GROUP.sup.a BIOPSIES.sup.b /TOTAL TOTAL TESTED

1 I 3/5 5/5 2 I 5/5 1/1 3 I 0/5 0/5 4 I 0/5 1/5 5 I 0/5 1/5 6 I 3/5 0/2 11/30 = 37% 8/23 = 35% 7 NI 5/5 4/5 8 NI 5/5 3/4 9 NI 5/5 4/5 10 NI 2/5 3/4 11 NI 4/5 4/5 21/25 = 84% 18/23 = 78%

I = Piglets fed diluted immune colostrum. NI = Piglets fed nonimmune preparation (Similac RTF). .sup.b Based upon colony growth from selective agar plates. H. pylori confirmed by appropriate biochemical testing (Urease, Catalase, Oxidase and sensitivity to nalidixic acid and cephalothin). .sup.c Gram stain of inoculum mucus from plates with or without typcial H pylori microcolonies (GNR = Gram Negative Rods).

Other Reference Publication (8):
Campylobacter Pylori, E. A. J. Rauws and G. N. J. Tytgat, editors, (1989), pp. 89-103 and 138-139.

CLAIMS:

1. A composition of matter having utility for providing passive immunity to a human when the composition is enterally ingested, said composition comprising non-denatured immunoglobulins which specifically bind to the bacterium Helicobacter pylori in the stomach of a human, said Helicobacter pylori being capable of colonizing and producing gastritis in humans and gnotobiotic piglets.

3. A method for producing a composition of matter having utility for providing passive immunity to a human when the composition is enterally ingested, said composition comprising immunoglobulins which specifically bind to the bacterium Helicobacter pylori, said method comprising the steps of:

(a) immunizing a lactating or pregnant cow with either a cell suspension or antigen derived from a strain of Helicobacter pylori capable of colonizing and producing gastritis in humans or gnotobiotic piglets;

(b) obtaining colostrum or milk from the cow; and

(c) isolating the immunoglobulins from said colostrum or milk.

First Hit Fwd Refs

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L1: Entry 18 of 18 File: USPT Nov 2, 1993

DOCUMENT-IDENTIFIER: US 5258178 A
**** See image for Certificate of Correction ****
TITLE: Method and product for the treatment of gastric disease

Abstract Text (1):
This invention describes a product obtained from the isolation and concentration of specific immunoglobulins (antibodies) derived from the mammary secretions of cows immunized with Helicobacter pylori. The product is useful in preparing formulations for the treatment and/or prevention of gastric diseases.

Brief Summary Text (2):
This invention relates to the isolation of specific immunoglobulins which, in the preferred embodiment, are isolated from the colostrum or milk of cows immunized with Helicobacter pylori. This invention is also directed to a method of use of these specific antibodies in the preparation of novel formulations useful in the enteral treatment of gastric disease.

Brief Summary Text (4):
The present invention relates to a process for the production of a protein concentrate containing immunological factors preferably of lactic origin and to a specific immunoglobulin population. More particularly, this invention relates to active immunoglobulins exhibiting specificity against the microorganism, Helicobacter pylori (formerly referred to in the literature as Campylobacter pylori) and the use of this protein in the management of Helicobacter pylori colonization in the gastrointestinal (GI) tract. More specifically, the present invention relates to the injection of lactating mammals with Helicobacter pylori, subsequent isolation and concentration of antibodies from the colostrum (or milk) produced by the immunized mammals and use of this concentrate, by way of enteral ingestion, in reducing the infectivity of Helicobacter pylori resident in the gastrointestinal tract. This protein (immunoglobulin) concentrate is useful in the treatment of pathological sequela associated with Helicobacter pylori colonization of the GI tract including gastritis and peptic ulcer disease.

Brief Summary Text (6):
U.S. Pat. Nos. 3,992,521 and 3,984,539 disclose a process for obtaining an immune product containing antibodies from the serum of a horse or cow and the immunoglobulin product itself. These patents do not suggest nor disclose the specific immunoglobulin of the present invention, nor its method of production and isolation, nor its intended method of use for the treatment of Helicobacter pylori induced gastritis.

Brief Summary Text (7):
U.S. Pat. No. 3,123,230 discloses a method for producing antibodies which consists of injecting a lactating mammal with a mixture of killed microorganisms and isolating the antibodies from serum or milk. This patent does not suggest nor disclose that Helicobacter pylori can induce an antibody response nor the specific method of treatment employed in this invention.

Brief Summary Text (9):
Ebina and colleagues disclose the immunization of cows with human rotavirus and the

isolation of immunoglobulin to the virus from the milk of cows. This immunoglobulin was orally administered to children and was found to reduce the frequency of the outbreak of diarrhea. See Ebina, et al., The Lancet (Oct. 29, 1983), 1029-1030, (1983); and Ebina, et al., Med. Microbiol. Immunol., 174, 177-185, (1985). These references do not suggest nor disclose that immunoglobulins to Helicobacter pylori would be useful in the management Helicobacter pylori induced gastritis.

Brief Summary Text (10):
U.S. Pat. No. 4,051,235 discloses the isolation of immunoglobulins from the milk of vaccinated cows by coagulating the milk, recovering the lactoserum (whey) and selectively precipitating the immunoglobulins with ammonium sulfate, followed by dialysis against water, filtration and drying. Seroprotection tests demonstrated that the protein concentrates of U.S. Pat. No. 4,051,231 provided local passive immunity in the intestine without resorption and without any significant loss of activity in the digestive tract, thereby providing generalized passive protection against certain enteropathogenic bacteria and/or viruses. This patent does not suggest nor disclose that such antibodies could serve to modify the course of Helicobacter pylori induced gastritis.

Brief Summary Text (11):
Much has been published regarding Helicobacter pylori itself. Helicobacter pylori is approximately 0.85 um in diameter with an average length of 2.9 um. The microorganism has a smooth coat and four to six polar flagella which are sheathed and have bulbous ends. In fresh cultures this organism appears as a slender, curved Gram-negative rod. Helicobacter pylori is readily distinguished from other gastric bacteria and spirochaetes by the absence of axial filaments in its flagella. Furthermore, optimum growth conditions for Helicobacter pylori are unusual and help to set it apart from other enteropathogens. For example, Helicobacter pylori requires a microaerophilic gas environment (i.e. low oxygen content) to sustain growth. Helicobacter pylori appears to tolerate a wide range of local pH conditions and is relatively resistant to acid conditions. It is believed that this resistance is due in part to the organism's outer protein structure which contains urease in large amounts resulting in the cleavage of urea naturally present in gastric fluid and hence, the formation of a buffering ammonia layer immediately around the organism.

Brief Summary Text (12):
Although a number of spiral bacteria inhabit the mouth and lower intestinal tract of all mammals, what distinguishes Helicobacter pylori is the is observation that it is localized almost exclusively to the luminal mucosal surface of the stomach and duodenum and generally is found deep within the gastric pits.

Brief Summary Text (13):
It is the combination of the unusual growth requirements and intestinal location which makes eradication and treatment of Helicobacter pylori so difficult. The ideal antimicrobial drug suitable for the successful treatment of Helicobacter pylori associated gastritis should exhibit local activity, be stable at low pH values and should be able to readily penetrate the gastric mucosa. These desirable properties of an antimicrobial are not easily accomplished and thus, satisfactory treatment of Helicobacter pylori with antimicrobials has yet to be accomplished.

Brief Summary Text (14):
The development of an agent which is effective in the management of Helicobacter pylori induced gastritis would fulfill a long felt need.

Brief Summary Text (15):
There is an emerging consensus in the field of gastroenterology that Helicobacter pylori is a major contributing-factor in the development of gastritis and peptic ulcer disease. Specifically, the following reference is useful in establishing the background of the present invention: Campylobacter pylori, E. A. J. Rauws and G. N.

J. Tytgat, editors, Adis Press Intntl. (1989).

Brief Summary Text (16):
In general, this reference discloses, at pages 138-139, the role of Helicobacter pylori in the development of gastritis and peptic ulcer disease. The key evidence in in support of Helicobacter pylori etiology in these conditions is based on the observation at pages 89-103, that elimination of Helicobacter pylori from the stomach through the use of antibiotics and/or bismuth compounds leads to a remission of the gastric disease.

Brief Summary Text (17):
Presently, the main therapies employed in the treatment of chronic active gastritis and peptic ulcer disease include the histamine H2-receptor antagonist's, bismuth compounds, and antibiotics. However, it is generally accepted that all currently used treatment modalitites are clinically inadequate since post-treatment relapse rates remain unacceptably high. In addition, several of these therapies are accompanied by significant side effects. For example, effective antibiotic treatment of Helicobacter pylori infections requires treatment over an extended duration (4-6 weeks) and results in the induction of diarrhea and intestinal discomfort. The bismuth compounds are also known to have a number of significant undesirable side effects.

Brief Summary Text (18):
To date, the preferred treatment has been dominated by the use of H2-antagonists which result in the suppression of acid and pepsin secretion; however, post treatment relapse rates are extremely high. Since symptoniatic relief and ulcer healing are the primary aim of treatment, without indefinite maintenance therapy, it is becoming increasingly apparent that a mucosal "protective agent" having antimicrobial activity against Helicobacter pylori, is desirable.

Brief Summary Text (19):
Thus, the medical community has a need for a protective agent which can be readily utilized in pharmaceutical and/or nutritional formulations. The present invention fulfills that need through the discovery that enteral ingestion of immunoglobulins derived from lactating mammals immunized with Helicobacter pylori provides such protection.

Brief Summary Text (20):
The prior art fails to suggest, disclose or contemplate the instant discovery which is, in part, the use of antibodies (immunoglobulin) in the treatment of Helicobacter pylori infection of the gastric mucosa and to the antibodies themselves.

Brief Summary Text (21):
Lactile secretion derived antibodies obtained from cows immunized with Helicobacter pylori will provide numerous advantages over other methods of immunoglobulin production. The advantages include quantity, ease and reproducibility of immunoglobulin isolation, ease of product preparation and significant cost savings as compared to antibody and product preparation based on other isolation methods.

Brief Summary Text (22):
One aspect of the present invention relates to a method for producing a milk based product having high immunological specific activity against Helicobacter pylori.

Brief Summary Text (24):
A further aspect of the present invention relates to a method for treating mammals in order to produce milk having immunological components which provide protection against Helicobacter pylori to subjects imbibing same.

Brief Summary Text (25):

A further aspect of this invention is the use of these specific antibodies (immunoglobulins) in the treatment of Helicobacter pylori induced gastritis.

Brief Summary Text (27):
There is disclosed a composition of matter consisting of non-denatured immunoglobulins which exhibit specific activity to the bacterium Helicobacter pylori. More specifically, an immunoglobulin isolated from the mammary secretions of of mammals exhibiting specific activity towards Helicobacter pylori .

Brief Summary Text (28):
There is also disclosed a medicament for gastritis caused by Helicobacter pylori which comprises non-denatured immunoglobulin which exhibits specificity towards Helicobacter pylori. The disclosed medicament may be used alone or in combination with a pharmacologically and/or nutritionally acceptable carrier and may be in a powdered or liquid form.

Brief Summary Text (29):
There is further disclosed a method for treating an individual suffering from Helicobacter pylori induced gastritis, peptic ulcer disease or other diseases said method consisting of administration to the individual in need of treatment an effective amount of a composition which contains at least the non-denatured immunoglobulins having specificity against Helicobacter pylor.

Brief Summary Text (30):
There is also disclosed a method for producing immunoglobulins exhibiting specificity for Helicobacter pylori which comprises the steps of 1) immunizing a lactating or pregnant mammal with a cell suspension of Helicobacter pylori emulsified in an adjuvant; 2) obtaining the colostrum or milk from the mammal; and 3) isolating the immunoglobulins from the secretion.

Brief Summary Text (31):
In general, the composition of matter of this invention is derived by a process which comprises the isolation of immunoglobulins from the mammary secretions of mammals immunized with Helicobacter pylori, said immunoglobulins exhibiting specific specific antimicrobial activity against Helicobacter pylori.

Brief Summary Text (32):
The method for the treatment of Helicobacter pylori infections, comprises the oral ingestion of an effective amount of Helicobacter pylori-specific immunoglobulins by a patient in need of treatment, said immunoglobulins being derived from the mammary secretions of mammals immunized with Helicobacter pylori. The immunoglobulins may be ingested alone or in combination with other materials such as fats, oils and proteins.

Brief Summary Text (33):
In its broadest aspect the present invention is directed to novel compositions which demonstrate antimicrobial activity against Helicobacter pylori.

Brief Summary Text (34):
The novel composition of matter of this invention consists of the immunoglobulins isolated from the mammary secretions of mammals immunized with Helicobacter pylori which may be utilized alone or combined with other natural or synthetic edible products such as lipids, proteins or oils. Said composition of matter is readily employed alone or in combination with other edible products to yield admixtures which are useful in the treatment of gastric diseases.

Brief Summary Text (37):
The utility of this invention was demonstrated by the ingestion of the specific antibody (immunoglobulins) of this invention by Helicobacter pylori infected germ free piglets. Ingestion of a nutritional containing the specific antibody

(immunoglobulins) of this invention provided the reduction or elimination of Helicobacter pylori induced pathology as well as a reduction in Helicobacter pylori bacterium colonization levels in various gastric epithelium regions, as determined by both agar plate culture reisolation and by histologic methods.

Brief Summary Text (38):
Specific antibody to Helicobacter pylori was raised in cows (as described in detail below) and characterized by standard immunochemical techniques as described below. Additional characterization of the antibodies can be achieved, for example, through the assessment of their ability to agglutinate Helicobacter pylori, their ability to fix complement in the presence of the Helicobacter pylori bacteria, the ability of the antibodies to inhibit bacterial replication and their ability to specifically bind the Helicobacter pylori bacterial antigens as detected by common immunochemical methods such as immunofluorescence and the like.

Brief Summary Text (39):
Following immunochemical characterization, the Helicobacter pylori specific antibodies were fed to Helicobacter pylori monoinfected gnotobiotic piglets according to the feeding regimen described below. Following feeding of Helicobacter pylori specific antibody, blood samples were drawn and the animals sacrificed for subsequent microbiological and histopathological assessment of the treatment protocol. The results of these studies were compared to Helicobacter pylori monoinfected gnotobiotic piglet littermates fed a nonimmune milk based on the nutritional product, Similac.RTM. (infant nutritional product of Ross Laboratories, Division of Abbott Laboratories, Columbus, Oh.) which does not contain specific Helicobacter pylori antibodies.

Brief Summary Text (40):
As a result of these experiments, the inventors have discovered that enteral ingestion of an the immunoglobulin product containing specific antibodies to Helicobacter pylori results in the reduction in the levels of viable Helicobacter pylori contained within various regions of the stomach and as such provides a realistic approach for the treatment of Helicobacter pylori induced gastritis. The Helicobacter pylori specific antibodies may be employed alone (i.e. in a liquid, tablet or capsule form) or in combination with other pharmaceutically acceptable carriers such as various lipids, is proteins or oils which may al so provide additional nutritional and/or pharmaceutical benefits.

Detailed Description Text (2):
The following examples relate to the production and use of specific antibodies to Helicobacter pylori and the physiological results of such usage. More specifically Examples 1 and 2 relate to the production and characterization of the immunoglobulin material isolated from pregnant cows immunized with Helicobacter pylori bacteria. Table 1 sets forth the immunological characterization of the colostrum whey products isolated from cows immunized with Helicobacter pylori as compared to non-immunized (i.e. control) cows. This data indicates that the concentration of Helicobacter pylori specific antibody (immunoglobulins) levels contained in whey provided by Helicobacter pylori immunized cows increased by over one hundred fold as compared to non-immunized cows. Example 3 relates to the biophysical and biological characterization of the specific immunoglobulins isolated from immunized versus non-immunized cows and exhibiting activity specifically against Helicobacter pylori. Tables 2 and 3 summarize the biological and biophysical data respectively.

Detailed Description Text (3):
Examples 4, 5 and 6 relate to the formulation and use of this immune material in the feeding of gnotobiotic pigs preinfected with Helicobacter pylori and thus, of the utility of this material in the treatment of Helicobacter pylori induced gastritis. The data contained in Table 4 indicates clearly that animals exposed to Helicobacter pylori through oral ingestion as described in Example 5 develop

systemic (sera contained) antibodies to Helicobacter pylori, thereby confirming the effectiveness of oral treatment with Helicobacter pylori as a means of achieving Helicobacter pylori infection.

Detailed Description Text (4):
Example 7 relates to the assessment of the effect of feeding this immune material on the levels of viable Helicobacter pylori bacteria which can subsequently be recovered from various gastric epithelial sites of piglets preinfected with Helicobacter pylori. Tables 5 and 6 summarize these results. The data indicate markedly reduced recoveries of viable Helicobacter pylori from all gastric regions examined for animals fed the immune product as compared to animals which received the nonimmune nutrient only. These results clearly indicate the effectiveness of using Helicobacter pylori specific antibodies (immunoglobulins) in the treatment of Helicobacter pylori induced gastritis.

Detailed Description Text (8):
Whey containing Helicobacter pylori specific antibodies was prepared from colostrum derived from a cow immunized while pregnant with whole formalin killed Helicobacter pylori bacteria (ATTCC Strain 26695). The bacteria, emulsified in incomplete Freund's adjuvant, were employed at a concentration of 5.times.10.sup.9 colony forming units (CFU)/mL. Each innoculation consisted of 12 mL of this material. The following immunization schedule was employed for each cow. Initially a subcutaneous (SQ) innoculation 14 days prior to drying off (D-14) was given. This was followed by an intramammary booster given seven days post drying off (D+7) and a second SQ booster given at D+30. This and similar immunization schedules are taught by the prior art and while the above schedule fully describes the method used, this description is not meant to limit the method of immunization under which the antibodies (immunoglobulins) to Helicobacter pylori can be raised since those skilled in the art will recognize and understand that other immunization methods would give similar results.

Detailed Description Text (14):
The bovine colostrum whey (BCW) samples were analyzed for total protein, immunoglobulin isotype type characterization, IgG1 content and for specific anti-Helicobacter pylori antibodies. The techniques used for these assays are standard procedures employed in the art and were, respectively, dye binding methods (BioRad), radial immunodiffusion, (ICN Biochemicals), and the enzyme linked immunoassay (ELISA).

Detailed Description Text (15):
The ELISA plates were coated with a Helicobacter pylori cell lysate at 3.2 ug/ml. The detecting antibody employed was a conjugate of alkaline phosphatase-coupled to a monoclonal antibody having immunospecificity for bovine IgG1 type antibodies. The indicator substrate for the assay was p-nitrophenylphosphate. The extent of color development was measured on a Dynatech ELISA plate reader at a visible wavelength of 490 nm and the data analyzed according to standard statistical methods. The results for these studies are summarized in Table 1. The data illustrates clearly that oral exposure to Helicobacter pylori results in over a one hundred fold increase in immunoglobulin (IgG1) concentration in colostral whey as compared to whey obtained from non-immunized cows.

Detailed Description Text (17):
Characterization of Helicobacter pylori Antibodies

Detailed Description Text (18):
Specific Helicobacter pylori antibodies may be characterized in a number of ways. For the purpose of the present study the biological activity of the immunoglobulins was determined by means of an agglutination assay (Table 2) and the biophysical characterization is provided by a consideration of the physical properties of the dominant immunoglobulin isotype contained in BCW (Table 3).

Detailed Description Text (20):
Test antibody dilutions were made in microtiter plates followed by the addition of appropriately diluted bacteria (formalin fixed Helicobacter pylori or E Coli). Buffer containing methylene blue was then added to facilitate easy reading of agglutination end poińts. The plates were examined after an eighteen (18) hour incubation at 4.degree. C. Thereafter, the extent of agglutination was determined and the results expressed as the lowest serial dilution of antibody which exhibited agglutination as defined above.

Detailed Description Text (21):
The results of this study are shown in Table 2. The data indicate clearly that antibodies raised against Helicobacter pylori and contained within BCW react specifically at high titers (i.e. dilutions) to Helicobacter pylori and do not react significantly with other viral and bacterial antigens. Likewise, similar immunoglobulin preparations from non-immunized is cows or cows immunized with unrelated bacterial or viral antigens do not react significantly with Helicobacter pylori.

Detailed Description Text (25):
Following assay, as described above, the BCW samples were added to commercially available Similac infant formula (Ross Laboratories) to yield a standard concentration of 17 mg IgGl/mL. Following combination the samples were heat treated and an antibiotic mixture added to result in the production of a material free of viable bacteria. This is a requirement for the gnotobiotic piglet model. The individual concentrations of the antibiotics were selected so as to be noninhibitory for Helicobacter pylori growth only. All other bacteria growth was inhibited.

Detailed Description Text (32):
Group II: 6 Piglets fed Similac a containing Helicobacter pylori antibody.

Detailed Description Text (33):
Both experimental groups were orally infected with Helicobacter pylori at 3 days of age with 2.times.10.sup.9 CFU/ML following pretreatment with Cimetidine.

Detailed Description Text (34):
Proof of Helicobacter pylori infection was established by means of ELISA assays to detect Helicobacter pylori-specific porcine antibodies in serum samples from each piglet as illustrated in Table 4. The data contained in this Table indicates that both groups of animals clearly develop significant antibody levels of all three immunoglobulin isotypes in their sera following infection as compared to preinfection sera.

Detailed Description Text (37):
Ten days following Helicobacter pylori infection, piglets were fed either Similac.RTM. only (Group I) or Similac.RTM. containing Helicobacter pylori specific antibodies (Group II) at a concentration of 17 mg IgGl/ml. The animals each received 30 mL of feed material three times each day. Following feeding, each animal received 150 mL of mild replacer diet which did not contain antibodies. The feeding protocol continued for a twenty day period.

Detailed Description Text (40):
At age 33 days, blood samples were drawn from each animal and the animals were euthanized and necropsied. The blood samples were processed to serum and analyzed for the presence of porcine anti-Helicobacter pylori antibodies. Gastric epithelium samples (1-2cm.sup.2) from five different anatomic regions were taken at necropsy and subsequently evaluated for bacterial colonization and histologic evidence of infection. The biopsies were taken from the cardiac, fundic, pyloric, antrum and diverticulum regions of the stomach.

Detailed Description Text (41):
Biopsy samples were placed on selective agar plates containing the selected antibiotic mixture and streaked. The innoculated plates were incubated in gas jars in a reduced oxygen atmosphere consisting of 5% O.sub.2, 10% CO.sub.2 and 80% N.sub.2 at 37.degree. C. which is a gas mixture which selectively facilitates Helicobacter pylori growth. The plates were examined for bacterial growth after 5-8 days. Suspected Helicobacter pylori colonies were subcultured onto fresh medium. Gram stains were performed on both the bacterial growth as well as on the mucoid material associated with the biopsies. Identity of the bacteria was confirmed using standard enzymatic (catalase, oxidase, urease) and antibiotic sensitivity (Nalidixic acid and Cephalothin) assays. The profiles provided by these assays allow for the accurate definition of the type of bacteria being examined. This methodology is standard in the art. The results for these studies are summarized in Tables 5 and 6.

Detailed Description Text (43):
In piglets fed non-immune nutrient, small bacterial colonies typical of Helicobacter pylori developed on the agar plates in 84% of the 5 stomach epithelium biopsy sites assayed. In comparison, piglets fed nutrient containing specific anti-Helicobacter pylori antibodies, colonies were observed in only 37% of the biopsies. Viable Helicobacter pylori bacteria were isolated from 100% of the control piglets (i.e. those animals receiving nonimmune nutrient) whereas viable Helicobacter pylori bacteria were isolated from only 50% of the piglets fed the immune product. Identity of the bacteria as being Helicobacter pylori was confirmed using biochemical assays. The differences are significant at the 95% confidence level using the one-sided Students "t" test or the nonparametric ranking approach (Wilcoxin test).

Detailed Description Text (44):
In addition to the above, Gram-stains were performed on the mucoid material isolated with the tissue biopsies following five days of incubation. (Helicobacter pylori is a Gram-negative bacterium). Specifically, a determination of the number of of biopsy specimens which were positive for Gram-negative bacteria was made. This information is summarized in Table 6. These data confirm the information contained in Table 5 in that the incidence of Gram-negative bacteria found in piglets fed the non-immune nutrient is 78% as compared to the 35% incidence found in piglets fed nutrient containing specific anti-Helicobacter pylori antibodies. This difference is significant at the 90% confidence level.

Detailed Description Text (45):
Of critical importance to the interpretation of the data discussed above is the determination that all experimental animals had indeed been infected with Helicobacter pylori bacteria. Proof of this is indicated by the data contained in Table 4 which illustrates that following infection with Helicobacter pylori all of the piglets developed antibodies to Helicobacter pylori (i.e. seroconvert) and contain antibodies (immunoglobulins) within their sera exhibiting activity specifically against Helicobacter pylori. This was determined by an ELISA analysis of the sera of the piglets upon termination of the experiment. The data contained in Table 4 indicates clearly that all animals developed significant amounts of specific anti-Helicobacter pylori antibodies and therefore must have been infected with the Helicobacter pylori microorganism.

Detailed Description Text (47):
The presence of Helicobacter pylori in the gastrointestinal tract of humans is believed to cause gastritis. Previous therapies have serious side effects and often fail to prevent reoccurance of the malady. The medical community has long sought a therapy or preventative to this disorder and the present invention fills that need.

Detailed Description Paragraph Table (1):
TABLE 1
CHARACTERIZATION OF BOVINE COLOSTRAL WHEY PREPARATION ELISA.sup.b COLOSTRAL WHEY TOTAL PROTEIN mg IgG1/ FOLD DIFFERENCE PREPARATION.sup.a (mg/mL) mL TITER/mg IgG1.sup.b FROM NON-IMMUNE
IMMUNE
176.2 125.2 615 123 CONTROL Not Determined 171.2 5 1 (Non-Immunized)
.sup.a
Colostral whey obtained from Helicobacter pylori immunized cow and from the nonHelicobacter pylori immunized cow (CONTROL). .sup.b Nonadjusted ELISA titers on undiluted colostral whey [IMMUNE = 77,000 and CONTROL = 860]. This assay detects antibodies specifically against Helicobacter pylori.

Detailed Description Paragraph Table (2):
TABLE 2
CHARACTERIZATION OF HELICOBACTER PYLORI (H. pylori) IMMUNE AND NON- IMMUNE BOVINE LACTOIMMUNOGLOBULIN PREPARATIONS BY BACTERIAL AGGLUTINATION.sup.a ANTIBODY TEST SAMPLE AGGLUTINATING ANTIBODY COLOSTRUM IMMUNIZING IgG1 TEST SPECIFIC ACTIVITY REFERENCE ANTIGEN (mg/mL) ANTIGEN TITER/mL (TITER/mgIgG1)
1 H. pylori 75.4 H. pylori 256,000 3,400 2 HRV.sup.b 52.4 H. pylori 2,560 49 3 E. coli 10 10 H. pylori 320 32 4 None 27.3 H. pylori 1,280 47 1 H. pylori 75.4 E. coli 640 8.5 2 HRV 52.4 E. coli 640 12 3 E. coli 10 E. coli 8,000 800 4 None 27.3 E. coli 160 5.9
.sup.a
H. pylori and E. coli bacteria were adjusted to 8 .times. 10.sup.9 cells/mL and added to twofold antibody dilutions in microtiter plates. Methylene blue (0.01%) was added to the bacterial diluent (0.01 M phosphate buffered saline pH 7.0) to enhance visualization of agglutinated bacteria. .sup.b Human rotavirus (HRV).

Detailed Description Paragraph Table (4):
TABLE 4
ISOTYPE SPECIFIC ELISA TESTING OF INFECTED AND CONTROL GERMFREE PIGLET SERA FOR ANTIBODY TO H. pylori MEAN OPTICAL DENSITY PIGLET GROUP ISOTYPE SPECIFIC SERUM ANTIBODY AND TIME SAMPLED IgG IgM IgA
ALL
PIGLETS <0.01 <0.02 <0.03 BEFORE INFECTION IMMUNE COLOSTRUM FED 0.58 0.32 0.38 AFTER INFECTION CONTROL (SIMILAC) FED 0.77 0.38 0.35 AFTER INFECTION

Detailed Description Paragraph Table (5):
TABLE 5
REISOLATION OF HELICOBACTER pylori FROM GERMFREE PIGLETS.sup.a PIGLET FEEDING H. pylori COLONY GROWTH.sup.c PIGLETS WITH H. pylori NUMBER GROUP.sup.b CARDIA FUNDUS PYLORUS ANTRUM DIVERTICULUM POSITIVE BIOPSIES/TOTAL.sup. d
1 I + - + + - 3/5 2 I + + + + + 5/5 3 I - - - - - 0/5 4 I - - - - - 0/5 5 I - - - - - 0/5 6 I + + - + - 3/5 11/30 = 37% 7 NI + + + + + 5/5 8 NI + + + + + 5/5 9 NI + + + + + 5/5 10 NI + - - - + 2/5 11 NI + - + + + 4/5 21/25 = 84%
.sup.a
Each piglet challenged with H. pylori (10 days earlier) was fed either H. pylori immune colostrum (1.5 g IgG1/90 mL/day in 3 feedings) diluted in Similac alone (nonimmune group) for 20 days. .sup.b I = Piglets fed diluted immune colostrum. NI = Piglets fed nonimmune preparation (Similac RTF). .sup.c Based upon colony growth from selective agar plates. H. pylori confirmed by appropriate biochemical testing (Urease, Catalase, Oxidase and sensitivity to nalidixic acid and cephalothin). .sup.d Statistical evaluation (2 sample, 1sided T test). Immune fed group significantly less than nonimmune group (p = 0.0298).

Detailed Description Paragraph Table (6):

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		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=AND	
☐	L1	700392 and (pylori or helicobacter or cpylori or hpylori or pyloridis or pyloris or pylorum)	3

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Search Results - Record(s) 1 through 3 of 3 returned.

☐ 1. 20040023983. 28 Aug 03. 05 Feb 04. Methods of use of fluoroquinolone compounds against pathogenic helicobacter bacteria. Chalker, Alison F., et al. 514/253.08; A61K031/496.

☐ 2. 20020168681. 20 Mar 01. 14 Nov 02. Microorganisms and assays for the identification of antibiotics. Yocum, R. Rogers, et al. 435/7.1; G01N033/53.

☐ 3. 20020107368. 06 Dec 00. 08 Aug 02. Helicobacter proteins, gene sequences and uses thereof. Tian, Jing-Hui, et al. 530/388.4; 424/190.1 530/350 536/23.7 A61K031/70 C07H021/04 C07K001/00 C07K014/00 C07K017/00 C07K016/00 C12P021/08.

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L1: Entry 1 of 3 File: PGPB Feb 5, 2004

PGPUB-DOCUMENT-NUMBER: 20040023983
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DOCUMENT-IDENTIFIER: US 20040023983 A1

TITLE: Methods of use of fluoroquinolone compounds against pathogenic helicobacter bacteria

PUBLICATION-DATE: February 5, 2004

INVENTOR-INFORMATION:

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ABSTRACT:

This invention relates, in part, to newly identified methods of using quinolone antibiotics, particularly a gemifloxacin compound against certain pathogenic bacteria.

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Set	Items	Description
		?s 26695 and (helicobacter? or pylori or pyloris or pyloridis) and (sds? or page? or electrophor? or kda or kd or kilodalton? or dalton? or rmw? or mw or size)
	116	26695
	19044	HELICOBACTER?
	19366	PYLORI
	10	PYLORIS
	178	PYLORIDIS
	39135	SDS?
	34285	PAGE?
	250906	ELECTROPHOR?
	94490	KDA
	45733	KD
	6701	KILODALTON?
	12966	DALTON?
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	8509	MW
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S1	25	26695 AND (HELICOBACTER? OR PYLORI OR PYLORIS OR PYLORIDIS) AND (SDS? OR PAGE? OR ELECTROPHOR? OR KDA OR KD OR KILODALTON? OR DALTON? OR RMW? OR MW OR SIZE)

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15750447 PMID: 14573621

Helicobacter pylori **heat shock protein 60 mediates interleukin-6 production by macrophages via a toll-like receptor (TLR)-2-, TLR-4-, and myeloid differentiation factor 88-independent mechanism.**

Gobert Alain P; Bambou Jean-Christophe; Werts Catherine; Balloy Viviane; Chignard Michel; Moran Anthony P; Ferrero Richard L

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Journal of biological chemistry (United States) Jan 2 2004, 279 (1) p245-50, ISSN 0021-9258 Journal Code: 2985121R

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Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Helicobacter pylori has been reported to induce interleukin-6 (IL-6) production in monocytes/macrophages and in chronically inflamed gastric tissues. The mechanism by which H. **pylori** induces IL-6 production in macrophages, however, has not been investigated. To identify the H. **pylori** factor responsible for this activity, we fractionated soluble proteins from H. **pylori** strain **26695** by ion exchange and **size** exclusion chromatography and screened the fractions for IL-6-inducing activity on RAW 264.7 macrophages. A single protein was purified and identified by mass spectrometry as H. **pylori** heat shock protein 60 (HSP60). Consistent with the observed IL-6-inducing activity of H. **pylori** HSP60, soluble protein extracts of H. **pylori** **26695** and SS1 strains that were depleted of this protein by affinity chromatography had dramatically reduced IL-6-inducing activities. The immunopurified HSP60 stimulated IL-6 production in macrophages. When stimulated with H. **pylori** HSP60 or intact bacteria, peritoneal macrophages from mice deficient in Toll-like receptor (TLR)-2, TLR-4, TLR-2/TLR-4, and myeloid differentiation factor 88 produced the same amount of IL-6 than macrophages from wild-type mice, demonstrating the independence of H. **pylori** HSP60 responses from these signaling molecules. H. **pylori** HSP60-induced IL-6 mRNA expression, and NF-kappaB activation in

RAW 264.7 cells was abrogated in the presence of MG-132, a proteasome inhibitor. In contrast, inhibitors of protein kinase A or C, mitogen-activated protein kinase kinase, and phosphoinositide 3-kinase had no effect on IL-6 mRNA levels. This study demonstrates the induction of innate immune responses by H. **pylori** HSP60, thereby implicating this highly conserved protein in the pathophysiology of chronic gastritis.

Tags: Support, Non-U.S. Gov't

Descriptors: Antigens, Differentiation--physiology--PH; *Chaperonin 60 --pharmacology--PD; * **Helicobacter pylori** ; *Interleukin-6--biosynthesis --BI; *Macrophages--immunology--IM; *Receptors, Immunologic--physiology--PH ; Animals; Base Sequence; Cell Differentiation; Cell Line; DNA Primers; Macrophages--cytology--CY; Macrophages--microbiology--MI; Mice; Mice, Knockout; Receptors, Immunologic--deficiency--DF; Reverse Transcriptase Polymerase Chain Reaction

CAS Registry No.: 0 (Antigens, Differentiation); 0 (Chaperonin 60); 0 (DNA Primers); 0 (Interleukin-6); 0 (MyD88 protein); 0 (Receptors, Immunologic)

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1/9/2

DIALOG(R)File 155:MEDLINE(R)

15719810 PMID: 14761989

Proteomic analysis of the sarcosine-insoluble outer membrane fraction of Helicobacter pylori **strain** 26695 .

Baik Seung-Chul; Kim Kyung-Mi; Song Su-Min; Kim Do-Su; Jun Jin-Su; Lee Seung-Gyu; Song Jae-Young; Park Jeong-Uck; Kang Hyung-Lyun; Lee Woo-Kon; Cho Myung-Je; Youn Hee-Shang; Ko Gyung-Hyuck; Rhee Kwang-Ho

Department of Microbiology, Gyeongsang Institute of Health Science, Gyeongsang National University College of Medicine, Jinju, Gyeongsangnam-do 660-751, Republic of Korea.

Journal of bacteriology (United States) Feb 2004, 186 (4) p949-55, ISSN 0021-9193 Journal Code: 2985120R

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Helicobacter pylori causes gastroduodenal disease, which is mediated in part by its outer membrane proteins (OMPs). To identify OMPs of H. **pylori** strain **26695** , we performed a proteomic analysis. A sarcosine-insoluble outer membrane fraction was resolved by two-dimensional **electrophoresis** with immobilized pH gradient strips. Most of the protein spots, with molecular masses of 10 to 100 **kDa** , were visible on the gel in the alkaline pI regions (6.0 to 10.0). The proteome of the OMPs was analyzed by matrix-assisted laser desorption ionization-time-of-flight mass spectrometry. Of the 80 protein spots processed, 62 spots were identified; they represented 35 genes, including 16 kinds of OMP. Moreover, we identified 9 immunoreactive proteins by immunoblot analysis. This study contributes to the characterization of the H. **pylori** strain **26695** proteome and may help to further elucidate the biological function of H. **pylori** OMPs and the pathogenesis of H. **pylori** infection.

Tags: Support, Non-U.S. Gov't

Descriptors: Bacterial Outer Membrane Proteins--analysis--AN; * **Helicobacter pylori** --chemistry--CH; *Proteomics; *Sarcosine --pharmacology--PD; Bacterial Outer Membrane Proteins--physiology--PH; **Electrophoresis** , Gel, Two-Dimensional; Hydrogen-Ion Concentration

CAS Registry No.: 0 (Bacterial Outer Membrane Proteins); 107-97-1 (Sarcosine)

Record Date Created: 20040205

Record Date Completed: 20040226

1/9/3
DIALOG(R)File 155:MEDLINE(R)

15512420 PMID: 14648714
Identification of candidate antigens for serologic detection of Helicobacter pylori **-infected patients with gastric carcinoma.**
Krah Alexander; Miehlke Stephan; Pleissner Klaus-Peter; Zimny-Arndt Ursula; Kirsch Christian; Lehn Norbert; Meyer Thomas F; Jungblut Peter R; Aebischer Toni
Department of Molecular Biology, Max Planck Institute for Infection Biology, Berlin, Germany.
International journal of cancer. Journal international du cancer (United States) Jan 20 2004, 108 (3) p456-63, ISSN 0020-7136 Journal Code: 0042124
Document type: Journal Article
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Helicobacter pylori colonizes the stomach of almost half the world population and is a causative agent of gastric carcinomas and duodenal ulcers. Only a small fraction of infected people will develop these severe illnesses and a predictive test to identify people at high risk would greatly benefit disease management. Our study aimed to identify conserved bacterial antigens that may be useful for the development of such a diagnostic test. High-resolution immunoproteomics by 2-dimensional **electrophoresis** of H. **pylori 26695** proteins was carried out with sera from infected patients with either duodenal ulcer (n=30) or gastric carcinoma (n=30), 2 clinically divergent conditions. According to their antigen recognition patterns clear groups of patients were identified. Although this classification did not correspond to the clinical status, it may be correlated to other bacterial or host factors that influence the outcome of infection. In general antigen recognition patterns were found to be highly variable, however by utilizing powerful image analysis and statistical tests the recognition of 14 antigenic protein species was found to differ significantly ($p<0.01$) between both diseases. Particular protein species of GroEL, HyuA, GroES and AtpA appear to be useful surrogate markers for gastric carcinoma detection and consequently should be considered for further prospective studies to assess their predictive value. For one protein species of AtpA, evidence was found that different post-translational modifications may confer different immunogenicities. Copyright 2003 Wiley-Liss, Inc.
Tags: Comparative Study; Female; Human; Male; Support, Non-U.S. Gov't
Descriptors: Adenocarcinoma--microbiology--MI; *Antigens, Bacterial --blood--BL; * **Helicobacter** Infections--microbiology--MI; * **Helicobacter pylori** --immunology--IM; *Stomach Neoplasms--microbiology--MI; Adenocarcinoma--pathology--PA; Adult; Aged; Aged, 80 and over; Bacterial Proteins--blood--BL; Duodenal Ulcer--microbiology--MI; Duodenal Ulcer --pathology--PA; **Electrophoresis** , Gel, Two-Dimensional; Immunoblotting; Middle Aged; Retrospective Studies; Spectrometry, Mass, Matrix-Assisted Laser Desorption-Ionization; Stomach Neoplasms--pathology--PA
CAS Registry No.: 0 (Antigens, Bacterial); 0 (Bacterial Proteins)
Record Date Created: 20031203
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1/9/4
DIALOG(R)File 155:MEDLINE(R)

15318174 PMID: 14519719
Analysis of Automatically Generated Peptide Mass Fingerprints of Cellular Proteins and Antigens from Helicobacter pylori 26695 **Separated by Two-dimensional** Electrophoresis .
Krah Alexander; Schmidt Frank; Becher Dorte; Schmid Monika; Albrecht Dirk ; Rack Axel; Buttner Knut; Jungblut Peter R

Core Facility for Protein Analysis and section sign Department of Molecular Biology, Max Planck Institute for Infection Biology, 10117 Berlin, and the ||Institute for Microbiology, Ernst Moritz Arndt University Greifswald, 17487 Greifswald, Germany.

Molecular & cellular proteomics - MCP (United States) Dec 2003, 2 (12) p1271-83, ISSN 1535-9476 Journal Code: 101125647
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Helicobacter pylori is a causative agent of severe diseases of the gastric tract ranging from chronic gastritis to gastric cancer. Cellular proteins of H. **pylori** were separated by high resolution two-dimensional gel **electrophoresis** . A dataset of 384 spots was automatically picked, digested, spotted, and analyzed by matrix-assisted laser desorption ionization mass spectrometry peptide mass fingerprint in triple replicates. This procedure resulted in 960 evaluable mass spectra. Using a new version of our data analysis software MS-Screener we improved identification and tested reliability of automatically generated data by comparing with manually produced data. Antigenic proteins from H. **pylori** are candidates for vaccines and diagnostic tests. Previous immunoproteomics studies of our group revealed antigen candidates, and 24 of them were now closely analyzed using the MS-Screener software. Only in three spots minor components were found that may have influenced their antigenicities. These findings affirm the value of immunoproteomics as a hypothesis-free approach. Additionally, the protein species distribution of the known antigen GroEL was investigated, dimers of the protein alkyl hydroperoxide reductase were found, and the fragmentation of gamma-glutamyltranspeptidase was demonstrated.

Record Date Created: 20031216
Date of Electronic Publication: 20030929

1/9/5

14276261 PMID: 10096092

Molecular genetic basis for the variable expression of Lewis Y antigen in Helicobacter pylori **: analysis of the alpha (1,2) fucosyltransferase gene.**

Wang G; Rasko D A; Sherburne R; Taylor D E
Department of Medical Microbiology and Immunology, University of Alberta, Edmonton, Canada.

Molecular microbiology (ENGLAND) Feb 1999, 31 (4) p1265-74, ISSN 0950-382X Journal Code: 8712028
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Helicobacter pylori lipopolysaccharides (LPS) express human oncofetal antigens Lewis X and Lewis Y. The synthesis of Lewis Y involves the actions of alpha (1,3) and alpha (1,2) fucosyltransferases (FucTs). Here, we report the molecular cloning and characterization of genes encoding H. **pylori** alpha (1,2) FucT (Hp fucT2) from various H. **pylori** strains. We constructed Hp fucT2 knock-out mutants and demonstrated the loss of Lewis Y production in these mutants by enzyme-linked immunosorbent assay (ELISA) and immunoelectron microscopy. The Hp fucT2 gene contains a hypermutable sequence [poly (C) and TAA repeats], which provides a possibility of frequent shifting into and out of coding frame by a polymerase slippage mechanism. Thus, the Hp fucT2 gene displays two major genotypes, consisting of either a single full-length open reading frame (ORF; as in the strain UA802) or truncated ORFs (as in the strain **26695**). In vitro expression of Hp fucT2 genes demonstrated that both types of the gene have the potential to produce the full-length protein. The production of the full-length

protein by the **26695** fucT2 gene could be attributed to translational-1 frameshifting, as a perfect translation frameshift cassette resembling that of the Escherichia coli dnaX gene is present. Examination of the strain UA1174 revealed that its fucT2 gene has a frameshifted ORF at the DNA level, which cannot be compensated by translation frameshifting, accounting for its Lewis Y off phenotype. In another strain, UA1218, the fucT2 gene is apparently turned off because of the loss of its promoter. Based on these data, we proposed a model for the variable expression of Lewis Y by H. **pylori** , in which regulation at the level of replication slippage (mutation), transcription and translation of the fucT2 gene may all be involved.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: Fucosyltransferases--genetics--GE; *Fucosyltransferases --metabolism--ME; * **Helicobacter pylori** --genetics--GE; *Lewis Blood-Group System--biosynthesis--BI; Amino Acid Sequence; Antigens, CD15 --biosynthesis--BI; Base Sequence; DNA, Bacterial--genetics--GE; **Electrophoresis** , Polyacrylamide Gel; Enzyme-Linked Immunosorbent Assay; Fucosyltransferases--chemistry--CH; **Helicobacter pylori** --enzymology--EN ; Immunoblotting; Microscopy, Immunoelectron; Molecular Sequence Data; Mutagenesis, Insertional; Plasmids--genetics--GE; Sequence Analysis, DNA

Molecular Sequence Databank No.: GENBANK/AF076779; GENBANK/AF093828; GENBANK/AF093829; GENBANK/AF093830; GENBANK/AF093831; GENBANK/AF093832; GENBANK/AF093833

CAS Registry No.: 0 (Antigens, CD15); 0 (DNA, Bacterial); 0 (Lewis Blood-Group System); 0 (Lewis Y antigen); 0 (Plasmids)

Enzyme No.: EC 2.4.1.- (Fucosyltransferases); EC 2.4.1.69 (galactoside 2-fucosyltransferase)

Record Date Created: 19990715

Record Date Completed: 19990715

1/9/6

DIALOG(R)File 155:MEDLINE(R)

13963265 PMID: 9663688

Natural competence for DNA transformation in Helicobacter pylori : **identification and genetic characterization of the comB locus.**

Hofreuter D; Odenbreit S; Henke G; Haas R

Max-Planck-Institut fur Biologie, Abteilung, Infektionsbiologie, Tubingen, Germany.

Molecular microbiology (ENGLAND) Jun 1998, 28 (5) p1027-38, ISSN 0950-382X Journal Code: 8712028

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The gram-negative bacterial pathogen **Helicobacter pylori** , an important aetiological agent of gastroduodenal disease in humans, belongs to a group of bacterial species displaying competence for genetic transformation. Here, we describe the comB gene locus of H. **pylori** involved in DNA transformation competence. It consists of a cluster of four tandemly arranged genes with partially overlapping open reading frames, orf2, comB1, comB2 and comB3, constituting a single transcriptional unit. Orf2 encodes a 37-amino-acid peptide carrying a signal sequence, whereas comB1, comB2 and comB3 produce 29 **kDa** , 38 **kDa** and 42 **kDa** proteins, respectively, as demonstrated by immunoblotting with specific antisera. For Orf2 and ComB1, no homologous proteins were identified in the database. For ComB3, the best homologies were found with TraS/TraB from the Pseudomonas aeruginosa conjugative plasmid RP1 and TrbI of plasmid RP4, VirB10 from the Ti plasmid of Agrobacterium tumefaciens and PtlG, a protein involved in secretion of pertussis toxin of Bordetella pertussis. Defined transposon knock-out mutants in individual comB genes resulted in transformation-defective phenotypes ranging from a 90% reduction to a complete loss of the natural transformation efficiency. The comB2 and comB3 genes show homology to HP0528 and HP0527, respectively, located on the

cagII pathogenicity island of H. **pylori** strain **26695** .
Descriptors: Bacterial Proteins--genetics--GE; *Chromosome Mapping; *DNA-Binding Proteins; *Genes, Bacterial; * **Helicobacter pylori** --genetics--GE; *Transformation, Bacterial; Amino Acid Sequence; Base Sequence; Chromosomes, Bacterial; DNA, Bacterial; Gene Expression; Molecular Sequence Data; Mutagenesis, Insertional; Operon; Sequence Analysis, DNA
Molecular Sequence Databank No.: GENBANK/AJ132366
CAS Registry No.: 0 (Bacterial Proteins); 0 (DNA, Bacterial); 0 (DNA-Binding Proteins); 127187-58-0 (comB protein, Haemophilus influenzae)
Record Date Created: 19980928
Record Date Completed: 19980928

1/9/7
DIALOG(R)File 155:MEDLINE(R)

13906239 PMID: 9604428
Helicobacter pylori **--molecular genetics and diagnostic typing.**
Ge Z; Taylor D E
Department of Medical Microbiology and Immunology, University of Alberta, Edmonton, Canada.
British medical bulletin (ENGLAND) 1998, 54 (1) p31-8, ISSN 0007-1420 Journal Code: 0376542
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
The genome of H. **pylori** is 1.68-1.73 Mb in **size** and contains a relatively low GC content (an average of 32.5 mol%). Physical and genetic maps of five H. **pylori** strains (NCTC 11637, NCTC 11638, NCTC 11639, UA 802 and UA 861) have been constructed and the complete genome sequence of strain **26695** has been determined. At least 50 genes, some of which play important roles in the physiology and pathogenicity of the bacterium, have been cloned. Marked genomic sequence variability has evolved from stain to strain demonstrated by random arrangement of 17 known genes on the chromosome and frequent mutations within individual genes. Based on such variability, sensitive and efficient molecular tying techniques such ribotyping, AR-PCR, PCR-RFLP, PCR-DNA sequencing and PFGE-RFLP have been developed and widely applied in both epidemiological and clinical studies of this pathogen. Subtypes of vacA (encoding a vacuolating cytotoxin) and the intermediate forms of a pathogenicity island (the cag region) have been identified in different H. **pylori** strains and these individual vacA subtypes are associated with specific clinical manifestations of H. **pylori** infection. Further studies on relationships between the genetic diversity and pathogenicity of H. **pylori** strains would lead to the development of novel and efficient therapeutic strategies for eradication of this microorganism. (23 Refs.)
Tags: Support, Non-U.S. Gov't
Descriptors: Genome, Bacterial; * **Helicobacter pylori** --genetics--GE; Bacterial Typing Techniques; Genetic Techniques; **Helicobacter pylori** --classification--CL
Record Date Created: 19980623
Record Date Completed: 19980623

1/9/8
DIALOG(R)File 155:MEDLINE(R)

12117908 PMID: 12445482
Expression and localization of alpha- and beta-carbonic anhydrase in Helicobacter pylori .
Chirica Laura C; Petersson Christoffer; Hurtig Marina; Jonsson

Bengt-Harald; Boren Thomas; Lindskog Sven
Department of Chemistry, Biochemistry, Umea University, SE-90187 Umea, Sweden.
Biochimica et biophysica acta (Netherlands) Dec 16 2002, 1601 (2) p192-9, ISSN 0006-3002 Journal Code: 0217513
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Helicobacter pylori , the causative agent of peptic ulcer disease, expresses two different forms of the zinc-containing enzyme carbonic anhydrase (CA) (alpha and beta), catalyzing the reversible hydration of CO(2). Presumably, the high CO(2) requirement of H. **pylori** implies an important role for this enzyme in the bacterial physiology. In this paper, expression of the CAs has been analyzed in three different strains of the bacterium, **26695** , J99 and 17.1, and appears to be independent of CO(2) concentration in the investigated range (0.1-10%). Presence of the potent and highly specific CA inhibitor, acetazolamide, in the medium does not seem to inhibit bacterial growth at the given sulfonamide concentration. Moreover, the localization and distribution of the alpha-CA was analyzed by immunonegative staining, while **SDS** -digested freeze-fracture immunogold labelling was used for the beta-form of the enzyme. The latter method has the advantage of allowing assessment of protein localization to distinct cell compartments and membrane structures. The resulting electron microscopy images indicate a localization of the beta-CA in the cytosol, on the cytosolic side of the inner membrane and on the outer membrane facing the periplasmic space. The alpha-enzyme was found attached to the surface of the bacterium.

Tags: Support, Non-U.S. Gov't
Descriptors: Carbonic Anhydrases--genetics--GE; * **Helicobacter pylori** --enzymology--EN; * **Helicobacter pylori** --genetics--GE; Carbonic Anhydrases--metabolism--ME; Freeze Fracturing; Gene Expression Regulation, Bacterial; Gene Expression Regulation, Enzymologic; Genome, Bacterial; **Helicobacter pylori** --ultrastructure--UL; Isoenzymes--genetics--GE; Isoenzymes--metabolism--ME
CAS Registry No.: 0 (Isoenzymes)
Enzyme No.: EC 4.2.1.1 (Carbonic Anhydrases)
Record Date Created: 20021126
Record Date Completed: 20021231

1/9/9
DIALOG(R)File 155:MEDLINE(R)

11925555 PMID: 12125208
Cloning and comparison of ten gene sequences of a Chilean H. pylori **strain with other H.** pylori **strains revealed higher variability for VacA and CagA virulence factors.**
Muller Ilse; Medina-Selby Angelica; Palacios Jose Luis; Martinez Patricio ; Opazo Patricio; Bruce Elsa; Mancilla Marta; Valenzuela Pablo; Yudelevich Arturo; Venegas Alejandro
BIOS Chile IGSA y MIFAB, Avda Marathon 1942, Santiago, Chile.
Biological research (Chile) 2002, 35 (1) p67-84, ISSN 0716-9760 Journal Code: 9308271
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

We have cloned and sequenced ten **Helicobacter pylori** genes from a Chilean strain (CH-CTX1) including: a cytotoxin VacA fragment, a CagA fragment (A17), a species-specific protein (TsaA), urease subunits (UreA, UreB), a flagellin subunit (FlaB), heat shock proteins (HspA and HspB), adhesin (HpaA) and a lipoprotein (Lpp20). We compared their deduced amino acid sequences with the corresponding sequences from three unrelated H.

pylori strains, including fully sequenced strains **26695** (UK) and J99(USA), and found that eight of them (UreA, UreB, FlaB, HspA, HspB, Lpp20, TsaA and HpaA) presented more than 97.3% identity. In contrast, VacA partial sequence showed lower identity values (93.2-94.9%). Moreover, we found major differences in the A17 region respect to the number and arrangement of the internal repeated elements when sequences from different strains were aligned. The A17 regions from strains CH-CTX1 and **26695** are very similar (91.8% identity) but lacked 6 repeated elements when compared to the Australian strains ATCC 43526 and NCTC 11637. The CCUG 17874 A17 region showed the largest deletion involving 9 repeats. A17 **size** differences between strains CCUG 17874 and CH-CTX1 were verified by PCR and polypeptide **size** . Such differences may explain variations in virulence among H. **pylori** strains as well as diversity in serum immunoreactivity.

Tags: Comparative Study; Support, Non-U.S. Gov't

Descriptors: Antigens, Bacterial; *Bacterial Proteins--genetics--GE; * **Helicobacter pylori** --genetics--GE; *Variation (Genetics); Alleles; Amino Acid Sequence; Base Sequence; Blotting, Western--methods--MT; Carrier Proteins--genetics--GE; Cloning, Molecular; DNA Primers; Genes; **Helicobacter pylori** --pathogenicity--PY; Molecular Sequence Data; Polymerase Chain Reaction--methods--MT; Virulence--genetics--GE

CAS Registry No.: 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (Carrier Proteins); 0 (DNA Primers); 0 (cagA protein, Helicobacter pylori); 0 (vacuolating toxin, Helicobacter pylori)

Record Date Created: 20020719

Record Date Completed: 20020904

1/9/10

DIALOG(R)File 155:MEDLINE(R)

11835699 PMID: 11981866

Identifying the major proteome components of Helicobacter pylori **strain** 26695 .

Cho Myung-Je; Jeon Beong-Sam; Park Jeong-Won; Jung Tae-Sung; Song Jae-Young; Lee Woo-Kon; Choi Yeo-Jeong; Choi Sang-Haeng; Park Seong-Gyu; Park Jeong-Uck; Choe Mi-Young; Jung Seun-Ae; Byun Eun-Young; Baik Seung-Chul; Youn Hee-Shang; Ko Gyung-Hyuck; Lim DongBin; Rhee Kwang-Ho

Department of Microbiology, College of Medicine, Gyeongsang National University, Chinju, Kyung-Nam, Korea.

Electrophoresis (Germany) Apr 2002, 23 (7-8) p1161-73, ISSN 0173-0835 Journal Code: 8204476

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The whole genome sequences of **Helicobacter pylori** strain **26695** have been reported. Whole cell proteins of H. **pylori** strain **26695** cells were obtained and analyzed by two-dimensional **electrophoresis** , using immobilized pH gradient strips. The most abundant proteins were shown in the region of pI 4.0-9.5 with molecular masses from 10 to 100 **kDa** . Soluble proteins were precipitated by the use of 0-80% saturated solutions of ammonium sulfate. Soluble proteins precipitated by the 0-40% saturations of ammonium sulfate produced similar spot profiles and their abundant protein spots had acidic pI regions. However, a number of soluble proteins precipitated by more than 60% saturation of ammonium sulfate were placed in the alkaline pI regions, compared to those precipitated by 40% saturation. In addition, we have performed an extensive proteome analysis of the strain utilizing peptide MALDI-TOF-MS. Among the 345 protein spots processed, 175 proteins were identified. The identified spots represented 137 genes. One-hundred and fifteen proteins were newly identified in this study, including DNA polymerase III beta-subunit. These results might provide guidance for the enrichment of H. **pylori** proteins and contribute to construct a master protein map of H. **pylori** .

Tags: Support, Non-U.S. Gov't

Descriptors: Bacterial Proteins--analysis--AN; * **Helicobacter pylori**

--chemistry--CH; *Proteome; **Electrophoresis** , Gel, Two-Dimensional; Molecular Weight; Spectrometry, Mass, Matrix-Assisted Laser Desorption-Ionization
CAS Registry No.: 0 (Bacterial Proteins); 0 (Proteome)
Record Date Created: 20020523
Record Date Completed: 20020918

1/9/11
DIALOG(R)File 155:MEDLINE(R)

11782176 PMID: 11966545
Clarithromycin increases the release of heat shock protein B from Helicobacter pylori .
Tsuzuki T; Ina K; Ohta M; Hasegawa T; Nagasaka T; Saburi N; Ueda M; Konagaya T; Kaneko H; Imada A; Nishiwaki T; Nobata K; Ando T; Kusugami K
First Department of Internal Medicine, Nagoya University School of Medicine, Showa-ku, Nagoya, Japan.
Alimentary pharmacology & therapeutics (England) Apr 2002, 16 Suppl 2 p217-28, ISSN 0269-2813 Journal Code: 8707234
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
BACKGROUND: Clarithromycin (CAM) may have certain indirect effects on **Helicobacter pylori** (H. **pylori**) other than its inhibitory activity on bacterial growth, as indicated in other infections with Gram-negative micro-organisms. In the present study, we examined the effects of lower concentrations of CAM on the release of heat shock protein B (HspB), one of the major antigenic proteins from H. **pylori** cells, as well as the changes in humoral immune response and histological degree of antral gastritis in patients who received eradication therapy with CAM. METHODS: The H. **pylori** strain **26695** and three CAM-resistant clinical isolates were cultured in broth with and without CAM (2-500 ng/mL). Expression of H. **pylori** proteins was examined by two-dimensional (2D)- **electrophoresis** followed by N-terminal amino acid sequencing. Changes in host immune response and histological degree of antral gastritis were monitored in patients with peptic ulcer disease who received H. **pylori** eradication therapy. RESULTS: 2D **electrophoresis** showed 26 spots in extracellularly released proteins with different profiles from those in cytoplasmic proteins. The release of HspB increased after incubation with CAM (30-500 ng/mL) in all three H. **pylori** clinical isolates tested. Patients with failed H. **pylori** eradication after triple therapy with CAM, but not those with failed eradication after dual therapy without CAM, showed an increase in serum IgG1 and IgG2 antibodies against HspB along with a decrease in the degree of neutrophil and H. **pylori** colonization density in tissue sections. CONCLUSIONS: CAM may induce a humoral immune response against H. **pylori** and a decrease in gastric mucosal inflammation through up-regulation of the release of HspB from the bacteria in infected patients.
Tags: Human
Descriptors: Anti-Bacterial Agents--therapeutic use--TU; *Antigens, Bacterial--biosynthesis--BI; *Clarithromycin--therapeutic use--TU; *Heat-Shock Proteins--biosynthesis--BI; * **Helicobacter** Infections--drug therapy--DT; * **Helicobacter** Infections--microbiology--MI; * **Helicobacter pylori** --isolation and purification--IP; Amino Acid Sequence; **Electrophoresis** , Gel, Two-Dimensional; **Helicobacter pylori** --immunology--IM; Molecular Sequence Data; Treatment Failure
CAS Registry No.: 0 (Anti-Bacterial Agents); 0 (Antigens, Bacterial); 0 (Heat-Shock Proteins); 0 (HspB protein); 81103-11-9 (Clarithromycin)
Record Date Created: 20020422
Record Date Completed: 20020715

1/9/12
DIALOG(R)File 155:MEDLINE(R)

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11743422 PMID: 11921447

Immunoproteomics of Helicobacter pylori **infection and relation to gastric disease.**

Haas Gaby; Karaali Galip; Ebermayer Karl; Metzger Wolfram G; Lamer Stephanie; Zimny-Arndt Ursula; Diescher Susanne; Goebel Ulf B; Vogt Konstanze; Roznowski Artur B; Wiedenmann Bertram J; Meyer Thomas F; Aebischer Toni; Jungblut Peter R

Department of Molecular Biology, Max Planck Institute for Infection Biology, Berlin, Germany.

Proteomics (Germany) Mar 2002, 2 (3) p313-24, ISSN 1615-9853 Journal Code: 101092707

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The Gram negative bacterium **Helicobacter pylori** is a human pathogen which infects the gastric mucosa and causes an inflammatory process leading to gastritis, ulceration and cancer. A systematic, proteome based approach was chosen to detect candidate antigens of H. **pylori** for diagnosis, therapy and vaccine development and to investigate potential associations between specific immune responses and manifestations of disease. Sera from patients with active H. **pylori** infection (n = 24), a control group with unrelated gastric disorders (n = 12) and from patients with gastric cancer (n = 6) were collected and analyzed for the reactivity against proteins of the strain HP **26695** separated by two-dimensional **electrophoresis** . Overall, 310 antigenic protein species were recognized by H. **pylori** positive sera representing about 17% of all spots separated. Out of the 32 antigens most frequently recognized by H. **pylori** positive sera, nine were newly identified and 23 were confirmed from other studies. Three newly identified antigens which belong to the 150 most abundant protein species of H. **pylori** , were specifically recognized by H. **pylori** positive sera: the predicted coding region HP0231, serine protease HtrA (HP1019) and Cag3 (HP0522). Other antigens were recognized differently by sera from gastritis and ulcer patients, which may identify them as candidate indicators for clinical manifestations. The data from these immunoproteomic analyses are added to our public database (http://www.mpiib-berlin.mpg.de/2D- **PAGE**). This platform enables one to compile many protein profiles and to integrate data from other studies, an approach which will greatly assist the search for more immunogenic proteins for diagnostic assays and vaccine design.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: Antigens, Bacterial--chemistry--CH; *Bacterial Proteins --chemistry--CH; * **Helicobacter** Infections--immunology--IM; * **Helicobacter pylori** --immunology--IM; *Proteome; *Stomach Diseases--immunology--IM; Antibodies, Bacterial--blood--BL; Antibodies, Bacterial--immunology--IM; Antigens, Bacterial--immunology--IM; Bacterial Proteins--immunology--IM; **Electrophoresis** , Gel, Two-Dimensional; **Helicobacter** Infections --microbiology--MI; **Helicobacter pylori** --chemistry--CH; **Helicobacter pylori** --genetics--GE; Immunoblotting; Peptide Mapping; Spectrometry, Mass, Matrix-Assisted Laser Desorption-Ionization; Stomach Diseases --microbiology--MI

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (Proteome)

Record Date Created: 20020328

Record Date Completed: 20020905

1/9/13

DIALOG(R)File 155:MEDLINE(R)

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11514401 PMID: 11681213

Tyrosine phosphorylation patterns and size **modification of the** Helicobacter pylori **CagA protein after translocation into gastric epithelial cells.**

Backert S; Muller E C; Jungblut P R; Meyer T F
Max-Planck-Institut fur Infektionsbiologie, Abt. Molekulare Biologie, Schumannstr. 20/21, D-10117 Berlin, Germany.
Proteomics (Germany) Apr 2001, 1 (4) p608-17, ISSN 1615-9853
Journal Code: 101092707
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Helicobacter pylori is one of the most common bacterial pathogens that causes a variety of gastric diseases. During infection, the immuno-dominant H. **pylori** CagA protein is translocated and tyrosine-phosphorylated in gastric epithelial cells. We compared tyrosine phosphorylation patterns of five CagA variants by two-dimensional **electrophoresis** (2-DE) and immunoblotting studies. Tyrosine-phosphorylated CagA was detected as two distinct protein species in strains P12, P227, G27 and **26695** suggesting that two tyrosine residues of CagA can be phosphorylated both separately and simultaneously. Prediction programs revealed the presence of three putative tyrosine phosphorylation motifs in the sequences of CagA. Mutations in these motifs were identified suggesting that only two putative phosphorylation-relevant tyrosines are present in each CagA variant. CagA of strain J99 was found to be unique because essential codons were mutated in each of the three motifs and, consequently, revealed no tyrosine phosphorylation signals at all. These findings support the view that CagA from different H. **pylori** strains can be tyrosine-phosphorylated at one or two out of three predicted positions. Additionally, truncated CagA protein species of about 100-105 **kDa** (p100CagA) have been detected after infection with some of the H. **pylori** strains. The isoelectric point determined by both 2-DE and sequence analysis suggested that p100CagA represents the amino (N)-terminal part of the protein. Translocation, tyrosine phosphorylation and **size** modification of CagA might be involved in host signal transduction and development of gastric disease.

Tags: Human; Support, Non-U.S. Gov't
Descriptors: Antigens, Bacterial; *Bacterial Proteins--chemistry--CH; *Bacterial Proteins--metabolism--ME; *Gastric Mucosa--metabolism--ME; *Gastric Mucosa--microbiology--MI; * **Helicobacter pylori** --metabolism --ME; Amino Acid Motifs; Amino Acid Sequence; Bacterial Proteins--genetics --GE; Base Sequence; Cell Line; Cloning, Molecular; DNA, Bacterial --genetics--GE; **Electrophoresis** , Gel, Two-Dimensional; Epithelial Cells --metabolism--ME; Genes, Bacterial; **Helicobacter pylori** --genetics--GE; **Helicobacter pylori** --pathogenicity--PY; Immunoblotting; Molecular Weight; Phosphorylation; Proteome; Spectrometry, Mass, Electrospray Ionization; Tyrosine--metabolism--ME
CAS Registry No.: 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (DNA, Bacterial); 0 (Proteome); 0 (cagA protein, Helicobacter pylori); 55520-40-6 (Tyrosine)
Record Date Created: 20011029
Record Date Completed: 20011218

1/9/14
DIALOG(R)File 155:MEDLINE(R)

11514393 PMID: 11681205
Identification of differentially regulated proteins in metronidozole resistant Helicobacter pylori **by proteome techniques.**
McAtee C P; Hoffman P S; Berg D E
Department of Applied Genomics, Bristol-Myers Squibb Pharmaceutical Research Institute, PO Box 5400, Princeton, NJ 08534-5400, USA. mcateep@bns.com
Proteomics (Germany) Apr 2001, 1 (4) p516-21, ISSN 1615-9853
Journal Code: 101092707
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Resistance to metronidazole (MTZ) is common among **Helicobacter pylori** strains in many societies, and results from loss of function mutations in genes for one or more cellular nitroreductases. When functional, these enzymes convert MTZ from a harmless prodrug to mutagenic and bacteriocidal products (probably hydroxylamine-type compounds), and in the process may generate active reactive oxygen metabolites. Here we examine the protein profiles of a derivative of strain **26695** that is resistant to moderate levels of MTZ because of mutation in rdxA (HP0954), the gene for the most important of these nitroreductases. The strain was grown with and without 18 micrograms/mL of MTZ to assess whether sublethal exposure triggers an adaptive response. Bacterial lysates were subjected to two-dimensional (2-D) **electrophoresis** and protein bands were identified by mass spectrometry and sequence analysis. Several proteins were decreased at least two-fold during growth with MTZ, yet the levels of various isoforms of alkylhydroperoxide reductase (AHP) (encoded by ahpC HP1563) were increased. AHP is an essential enzyme, and had been linked to resistance to oxygen toxicity in various prokaryotic and eukaryotic systems; we propose that the ability of an rdxA mutant strain to increase AHP abundance during exposure to MTZ is critically important in the realization of the resistance phenotype. More generally, these results highlight the potential of proteome analysis to tracing out how pathogenic bacteria cope with the challenges imposed on them by therapy or host responses to infection.

Tags: Human

Descriptors: Bacterial Proteins--isolation and purification--IP; * **Helicobacter pylori** --chemistry--CH; * **Helicobacter pylori** --drug effects--DE; Bacterial Proteins--genetics--GE; Drug Resistance, Bacterial; **Electrophoresis** , Gel, Two-Dimensional; Gene Expression Regulation, Bacterial; **Helicobacter** Infections--drug therapy--DT; **Helicobacter** Infections--microbiology--MI; **Helicobacter pylori** --genetics--GE; **Helicobacter pylori** --pathogenicity--PY; Metronidazole--pharmacology--PD ; Peptide Mapping; Proteome--genetics--GE; Proteome --isolation and purification--IP; Spectrometry, Mass, Matrix-Assisted Laser Desorption-Ionization; Virulence

CAS Registry No.: 0 (Bacterial Proteins); 0 (Proteome); 443-48-1 (Metronidazole)

Record Date Created: 20011029
Record Date Completed: 20011218

1/9/15
DIALOG(R)File 155:MEDLINE(R)

11500277 PMID: 11605832

Bioinformatics and mass spectrometry for microorganism identification: proteome-wide post-translational modifications and database search algorithms for characterization of intact H. pylori .

Demirev P A; Lin J S; Pineda F J; Fenselaut C

Department of Chemistry, University of Maryland, College Park 20742, USA. demirev@wam.umd.edu

Analytical chemistry (United States) Oct 1 2001, 73 (19) p4566-73, ISSN 0003-2700 Journal Code: 0370536

Comment in Anal Chem. 2001 Oct 1;73(19) 531A; Comment in PMID 11605859

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

MALDI-TOF mass spectrometry has been coupled with Internet-based proteome database search algorithms in an approach for direct microorganism identification. This approach is applied here to characterize intact H. **pylori** (strain **26695**) Gram-negative bacteria, the most ubiquitous human pathogen. A procedure for including a specific and common posttranslational modification, N-terminal Met cleavage, in the search algorithm is

described. Accounting for posttranslational modifications in putative protein biomarkers improves the identification reliability by at least an order of magnitude. The influence of other factors, such as number of detected biomarker peaks, proteome **size** , spectral calibration, and mass accuracy, on the microorganism identification success rate is illustrated as well.

Tags: Support, U.S. Gov't, Non-P.H.S.

Descriptors: Algorithms; *Bacterial Proteins--metabolism--ME; *Computational Biology--methods--MT; * **Helicobacter pylori** --metabolism --ME; *Protein Processing, Post-Translational; *Proteome--metabolism--ME; *Spectrometry, Mass, Matrix-Assisted Laser Desorption-Ionization--methods --MT; Bacterial Proteins--analysis--AN; Databases, Factual; **Helicobacter pylori** --chemistry--CH; **Helicobacter pylori** --classification--CL

CAS Registry No.: 0 (Bacterial Proteins); 0 (Proteome)

Record Date Created: 20011018

Record Date Completed: 20011204

1/9/16

DIALOG(R)File 155:MEDLINE(R)

11429210 PMID: 11526127

Differentiation of metronidazole-sensitive and -resistant clinical isolates of Helicobacter pylori **by immunoblotting with antisera to the RdxA protein.**

Latham S R; Owen R J; Elviss N C; Labigne A; Jenks P J

Department of Medical Microbiology, Royal Free and University College Medical School, London, United Kingdom.

Journal of clinical microbiology (United States) Sep 2001, 39 (9) p3052-5, ISSN 0095-1137 Journal Code: 7505564

Document type: Evaluation Studies; Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Antimicrobial resistance in **Helicobacter pylori** is a serious and increasing problem, and the development of rapid, reliable methods for detecting resistance would greatly improve the selection of antibiotics used to treat gastric infection with this organism. We assessed whether detection of the RdxA protein could provide the basis for determining the susceptibility of H. **pylori** to metronidazole. In order to raise polyclonal antisera to RdxA, we cloned the rdxA gene from H. **pylori** strain **26695** into the commercial expression vector pMAL-c2, purified the resultant fusion protein by affinity chromatography, and used this recombinant RdxA preparation to immunize rabbits. We then used this specific anti-RdxA antibody to perform immunoblotting on whole bacterial cell lysates of 17 metronidazole-sensitive and 27 metronidazole-resistant clinical isolates of H. **pylori** . While a 24- **kDa** immunoreactive band corresponding to the RdxA protein was observed in all metronidazole-sensitive strains, this band was absent in 25 of 27 resistant isolates. Our results indicate that testing for the absence of the RdxA protein would identify the majority of clinical isolates that will respond poorly to metronidazole-containing eradication regimens and have implications for the development of assays capable of detecting metronidazole resistance in H. **pylori** .

Tags: Human; Support, Non-U.S. Gov't

Descriptors: Anti-Bacterial Agents--pharmacology--PD; *Antibodies, Bacterial--immunology--IM; *Bacterial Proteins--immunology--IM; * **Helicobacter pylori** --classification--CL; * **Helicobacter pylori** --drug effects--DE; *Membrane Proteins--immunology--IM; *Metronidazole --pharmacology--PD; Animals; Drug Resistance, Bacterial; **Helicobacter** Infections--microbiology--MI; Immunoblotting; Rabbits

CAS Registry No.: 0 (Anti-Bacterial Agents); 0 (Antibodies, Bacterial) ; 0 (Bacterial Proteins); 0 (Membrane Proteins); 148711-16-4 (RdxA protein, Rhodobacter sphaeroides); 443-48-1 (Metronidazole)

Record Date Created: 20010829

Record Date Completed: 20011204

1/9/17
DIALOG(R)File 155:MEDLINE(R)

11410377 PMID: 11505637
Organization of the Helicobacter pylori **genome]**
Organizacja genomu **Helicobacter pylori** .
Zawilak A; Zakrzewska-Czerwinska J
Laboratorium Biologii Molekularnej Mikroorganizmow Instytutu Immunologii i Terapii Doswiadczalnej PAN im. L. Hirszfelda we Wroclawiu.
Postepy higieny i medycyny doswiadczalnej (Poland) 2001, 55 (3) p355-67, ISSN 0032-5449 Journal Code: 0421052
Document type: Journal Article; Review; Review, Tutorial ; English Abstract
Languages: POLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Helicobacter pylori is a Gram-negative, spiral-shaped pathogenic bacterium that was firstly isolated and cultured from biopsy specimens by Marshall and Warren in 1983. This organism is a human gastric pathogen associated with peptic ulcer disease as well as chronic gastritis. Recent epidemiological studies have demonstrated that H. **pylori** is a primary risk factor for the development of intestinal type gastric adenocarcinoma. H. **pylori** is the first bacterium for which the genomes of two unrelated strains (**26695** and J99) have been sequenced. The genome of H. **pylori** is relatively low in **size** (1.6-1.73 Mb). In this review, we compare the organization of two sequenced H. **pylori** genomes. A special emphasis on genetic diversity of H. **pylori** including plasticity zone and cag pathogenicity island has been placed. (33 Refs.)
Tags: Human; Support, Non-U.S. Gov't
Descriptors: Antigens, Bacterial; * **Helicobacter pylori** --genetics--GE ; Bacterial Proteins--metabolism--ME; Chromosome Mapping; Chronic Disease; DNA Replication; DNA, Bacterial--metabolism--ME; Gastritis--microbiology --MI; Genome; **Helicobacter pylori** --classification--CL; **Helicobacter pylori** --pathogenicity--PY; Peptic Ulcer--microbiology--MI; RNA, Bacterial --metabolism--ME; Restriction Mapping; Species Specificity; Stomach Neoplasms--microbiology--MI; Transcription, Genetic; Variation (Genetics)
CAS Registry No.: 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (DNA, Bacterial); 0 (RNA, Bacterial); 0 (cagA protein, Helicobacter pylori)
Record Date Created: 20010816
Record Date Completed: 20011207

1/9/18
DIALOG(R)File 155:MEDLINE(R)

11177765 PMID: 11179990
pH-dependent protein profiles of Helicobacter pylori **analyzed by two-dimensional gels.**
Slonczewski J L; McGee D J; Phillips J; Kirkpatrick C; Mobley H L
Department of Biology, Kenyon College, Gambier, OH 43022, USA. slonczewki@kenyon.edu
Helicobacter (United States) Dec 2000, 5 (4) p240-7, ISSN 1083-4389 Journal Code: 9605411
Contract/Grant No.: AI10098-01; AI; NIAID; AI25567; AI; NIAID
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
BACKGROUND: **Helicobacter pylori** survives transient exposure to

extreme acid prior to adherence and growth on the gastric epithelium at neutral pH. MATERIALS AND METHODS: The effect of pH stress on protein profiles of H. **pylori** was observed using two-dimensional gel **electrophoresis** (2-D gels). H. **pylori** **26695** was grown microaerobically in tryptone-yeast extract broth, 3% fetal bovine serum. Growth in acid alkalinized the medium, whereas growth in base caused acidification. For 2-D gel analysis of protein profiles, cultures were grown in media buffered at pH 5.7 and at pH 7.5. RESULTS: Under all pH conditions, the most abundant proteins observed were the urease structural subunit UreB and the chaperonin GroEL. Growth in acid significantly increased the abundance of UreB. Thus, urease expression is not completely constitutive, as reported previously, but shows regulation by pH. Another protein observed only at low pH was identified as mammalian apolipoprotein A-I, possibly taken up by H. **pylori** from bovine serum in the growth medium. This finding, if confirmed, suggests that uptake of high-density lipoprotein from the human host may facilitate acquisition of cholesterol, required for formation of the unique cholesteryl glucosides in the membrane of H. **pylori** . In growth above pH 7, three stress proteins were induced: GroES (HspA), GroEL (HspB), and the antioxidant AhpC homolog TsaA. In addition, N-terminal sequence analysis identified five additional proteins that had not previously been reported on 2-D gels of H. **pylori** (FMN, SodB, TrxB, TsaA, and Tsr). CONCLUSIONS: In summary, our 2-D gel study reveals expression of several proteins dependent on growth pH.

Tags: Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: Bacterial Proteins--analysis--AN; * **Helicobacter pylori** --chemistry--CH; * **Helicobacter pylori** --growth and development--GD; Culture Media; **Electrophoresis** , Gel, Two-Dimensional; Hydrogen-Ion Concentration

CAS Registry No.: 0 (Bacterial Proteins); 0 (Culture Media)

Record Date Created: 20010222

Record Date Completed: 20010621

1/9/19

DIALOG(R)File 155:MEDLINE(R)

11083503 PMID: 11120683

Finding pathogenicity islands and gene transfer events in genome data.

Lio P; Vannucci M

Department of Zoology, University of Cambridge, Downing Street, Cambridge CB2 3EH, UK. p.lio@zoo.cam.ac.uk

Bioinformatics (Oxford, England) (ENGLAND) Oct 2000, 16 (10) p932-40 , ISSN 1367-4803 Journal Code: 9808944

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

MOTIVATION: There is a growing literature on wavelet theory and wavelet methods showing improvements on more classical techniques, especially in the contexts of smoothing and extraction of fundamental components of signals. G+C patterns occur at different lengths (scales) and, for this reason, G+C plots are usually difficult to interpret. Current methods for genome analysis choose a window **size** and compute a chi(2) statistics of the average value for each window with respect to the whole genome. RESULTS: Firstly, wavelets are used to smooth G+C profiles to locate characteristic patterns in genome sequences. The method we use is based on performing a chi(2) statistics on the wavelet coefficients of a profile; thus we do not need to choose a fixed window **size** , in that the smoothing occurs at a set of different scales. Secondly, a wavelet scalogram is used as a measure for sequence profile comparison; this tool is very general and can be applied to other sequence profiles commonly used in genome analysis. We show applications to the analysis of Deinococcus radiodurans chromosome I, of two strains of **Helicobacter pylori** (**26695** , J99) and two of Neisseria meningitidis (serogroup B strain MC58 and serogroup A strain Z2491). We report a list of loci that have different G+C content with

respect to the nearby regions; the analysis of N. meningitidis serogroup B shows two new large regions with low G+C content that are putative pathogenicity islands. AVAILABILITY: Software and numerical results (profiles, scalograms, high and low frequency components) for all the genome sequences analyzed are available upon request from the authors.

Tags: Support, Non-U.S. Gov't

Descriptors: Gene Transfer, Horizontal--genetics--GE; *Genome, Bacterial; * **Helicobacter pylori** --genetics--GE; *Neisseria meningitidis--genetics --GE; Base Composition; Base Sequence; DNA, Bacterial; Data Interpretation, Statistical; Databases, Factual; GC Rich Sequence; **Helicobacter pylori** --pathogenicity--PY; Models, Genetic; Models, Statistical; Neisseria meningitidis--pathogenicity--PY; Numerical Analysis, Computer-Assisted; Sequence Analysis, DNA

CAS Registry No.: 0 (DNA, Bacterial)

Record Date Created: 20010116

Record Date Completed: 20010405

1/9/20

DIALOG(R)File 155:MEDLINE(R)

10737777 PMID: 10858232

Comparative genomics of Helicobacter pylori : **analysis of the outer membrane protein families.**

Alm R A; Bina J; Andrews B M; Doig P; Hancock R E; Trust T J

Infection Discovery AstraZeneca R & D Boston, Waltham, Massachusetts 02451, USA. richard.alm@astrazeneca.com

Infection and immunity (UNITED STATES) Jul 2000, 68 (7) p4155-68, ISSN 0019-9567 Journal Code: 0246127

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The two complete genomic sequences of **Helicobacter pylori** J99 and **26695** were used to compare the paralogous families (related genes within one genome, likely to have related function) of genes predicted to encode outer membrane proteins which were present in each strain. We identified five paralogous gene families ranging in **size** from 3 to 33 members; two of these families contained members specific for either H. **pylori** J99 or H. **pylori** **26695** . Most orthologous protein pairs (equivalent genes between two genomes, same function) shared considerable identity between the two strains. The unusual set of outer membrane proteins and the specialized outer membrane may be a reflection of the adaptation of H. **pylori** to the unique gastric environment where it is found. One subfamily of proteins, which contains both channel-forming and adhesin molecules, is extremely highly related at the sequence level and has likely arisen due to ancestral gene duplication. In addition, the largest paralogous family contained two essentially identical pairs of genes in both strains. The presence and genomic organization of these two pairs of duplicated genes were analyzed in a panel of independent H. **pylori** isolates. While one pair was present in every strain examined, one allele of the other pair appeared partially deleted in several isolates.

Tags: Comparative Study; Human

Descriptors: Bacterial Outer Membrane Proteins--genetics--GE; *Genome, Bacterial; * **Helicobacter pylori** --genetics--GE; Alleles; Amino Acid Sequence; Base Sequence; Binding Sites--genetics--GE; Conserved Sequence; DNA Primers--genetics--GE; Evolution, Molecular; Gene Duplication; **Helicobacter pylori** --classification--CL; **Helicobacter pylori** --metabolism--ME; Molecular Sequence Data; Multigene Family; Phylogeny; Protein Sorting Signals--genetics--GE; Ribosomes--metabolism--ME; Sequence Homology, Amino Acid; Species Specificity

CAS Registry No.: 0 (Bacterial Outer Membrane Proteins); 0 (DNA Primers); 0 (Protein Sorting Signals)

Record Date Created: 20000720

Record Date Completed: 20000720

1/9/21
DIALOG(R)File 155:MEDLINE(R)

10727728 PMID: 10844659
Comparative proteome analysis of Helicobacter pylori .
Jungblut P R; Bumann D; Haas G; Zimny-Arndt U; Holland P; Lamer S; Siejak F; Aebischer A; Meyer T F
Max-Planck-Institute for Infection Biology, Central Support Unit Biochemistry, Berlin, Germany. jungblut@mpiib-berlin.mpg.de
Molecular microbiology (ENGLAND) May 2000, 36 (3) p710-25, ISSN 0950-382X Journal Code: 8712028
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Helicobacter pylori , the causative agent of gastritis, ulcer and stomach carcinoma, infects approximately half of the worlds population. After sequencing the complete genome of two strains, **26695** and J99, we have approached the demanding task of investigating the functional part of the genetic information containing macromolecules, the proteome. The proteins of three strains of H. **pylori** , **26695** and J99, and a prominent strain used in animal models SS1, were separated by a high-resolution two-dimensional **electrophoresis** technique with a resolution power of 5000 protein spots. Up to 1800 protein species were separated from H. **pylori** which had been cultivated for 5 days on agar plates. Using matrix-assisted laser desorption/ionization mass spectrometry (MALDI-MS) peptide mass fingerprinting we have identified 152 proteins, including nine known virulence factors and 28 antigens. The three strains investigated had only a few protein spots in common. We observe that proteins with an amino acid exchange resulting in a net change of only one charge are shifted in the two-dimensional **electrophoresis** (2-DE) pattern. The expression of 27 predicted conserved hypothetical open reading frames (ORFs) and six unknown ORFs were confirmed. The growth conditions of the bacteria were shown to have an effect on the presence of certain proteins. A preliminary immunoblotting study using human sera revealed that this approach is ideal for identifying proteins of diagnostic or therapeutic value. H. **pylori** 2-DE patterns with their identified protein species were added to the dynamic 2D- **PAGE** database (http://www.mpiib-berlin.mpg.de/2D- **PAGE** /). This basic knowledge of the proteome in the public domain will be an effective instrument for the identification of new virulence or pathogenic factors, and antigens of potentially diagnostic or curative value against H. **pylori** .
Tags: Human
Descriptors: Bacterial Proteins--analysis--AN; * **Helicobacter pylori** --chemistry--CH; *Proteome--analysis--AN; Bacterial Proteins--chemistry--CH ; Databases, Factual; **Electrophoresis** , Gel, Two-Dimensional; **Helicobacter pylori** --genetics--GE; **Helicobacter pylori** --growth and development--GD; Hydrogen-Ion Concentration; Immunoblotting; Molecular Weight; Open Reading Frames; Proteome--chemistry--CH; Spectrometry, Mass, Matrix-Assisted Laser Desorption-Ionization
CAS Registry No.: 0 (Bacterial Proteins); 0 (Proteome)
Record Date Created: 20000829
Record Date Completed: 20000829

1/9/22
DIALOG(R)File 155:MEDLINE(R)

10701932 PMID: 10816463
Intercellular communication in Helicobacter pylori : **luxS is essential for the production of an extracellular signaling molecule.**
Forsyth M H; Cover T L

Departments of Medicine and Microbiology and Immunology, Vanderbilt University School of Medicine, Nashville, Tennessee, USA.

Infection and immunity (UNITED STATES) Jun 2000, 68 (6) p3193-9, ISSN 0019-9567 Journal Code: 0246127
Contract/Grant No.: AI 39657; AI; NIAID
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Individual bacteria of numerous species can communicate and coordinate their actions via the production, release, and detection of extracellular signaling molecules. In this study, we used the Vibrio harveyi luminescence bioassay to determine whether **Helicobacter pylori** produces such a factor. Cell-free conditioned media from H. **pylori** strains 60190 and **26695** each induced >100-fold-greater luminescence in V. harveyi than did sterile culture medium. The H. **pylori** signaling molecule had a molecular mass of <10 **kDa** , and its activity was unaffected by heating to 80 degrees C for 5 min or protease treatment. The genome sequence of H. **pylori** **26695** does not contain any gene predicted to encode an acyl homoserine lactone synthase but does contain an orthologue of luxS, which is required for production of autoinducer-2 (AI-2) in V. harveyi. To evaluate the role of luxS in H. **pylori** , we constructed luxS null mutants derived from H. **pylori** 60190 and **26695** . Conditioned media from the wild-type H. **pylori** strains induced >100-fold-greater luminescence in the V. harveyi bioassay than did conditioned medium from either mutant strain. Production of the signaling molecule was restored in an H. **pylori** luxS null mutant strain by complementation with a single intact copy of luxS placed in a heterologous site on the chromosome. In addition, Escherichia coli DH5alpha produced autoinducer activity following the introduction of an intact copy of luxS from H. **pylori** . Production of the signaling molecule by H. **pylori** was growth phase dependent, with maximal production occurring in the mid-exponential phase of growth. Transcription of H. **pylori** vacA also was growth phase dependent, but this phenomenon was not dependent on luxS activity. These data indicate that H. **pylori** produces an extracellular signaling molecule related to AI-2 from V. harveyi. We speculate that this signaling molecule may play a role in regulating H. **pylori** gene expression.

Tags: Comparative Study; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

Descriptors: Bacterial Proteins--genetics--GE; * **Helicobacter pylori** --physiology--PH; Amino Acid Sequence; Bacterial Proteins--biosynthesis--BI ; Biological Assay; Chemotactic Factors--metabolism--ME; Culture Media, Conditioned; Cytotoxins--biosynthesis--BI; Escherichia coli--genetics--GE; Gene Expression Regulation, Bacterial; Genetic Complementation Test; **Helicobacter pylori** --pathogenicity--PY; Homoserine --analogs and derivatives--AA; Homoserine--metabolism--ME; Lactones--metabolism--ME; Molecular Sequence Data; Mutation; Recombinant Proteins; Sequence Alignment ; Signal Transduction--genetics--GE; Vibrio--genetics--GE

CAS Registry No.: 0 (Bacterial Proteins); 0 (Chemotactic Factors); 0 (Culture Media, Conditioned); 0 (Cytotoxins); 0 (Lactones); 0 (LuxS protein); 0 (N-octanoylhomoserine lactone); 0 (Recombinant Proteins); 0 (vacuolating toxin, Helicobacter pylori); 498-19-1 (Homoserine)

Record Date Created: 20000623
Record Date Completed: 20000623

1/9/23

10577413 PMID: 10682319

Analysis of the genetic diversity of Helicobacter pylori : **the tale of two genomes.**

Alm R A; Trust T J
AstraZeneca R&D Boston, Cambridge, MA 02139-4239, USA. richard.alm@astrazeneca.com

Journal of molecular medicine (Berlin, Germany) (GERMANY) Dec 1999, 77 (12) p834-46, ISSN 0946-2716 Journal Code: 9504370
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Infection with **Helicobacter pylori** has been linked to numerous severe gastroduodenal diseases including peptic ulcer and gastric cancer. Several techniques have been used to measure the genetic heterogeneity of H. **pylori** at several different levels and to determine whether there is any correlation with severity of disease. The availability of two completed genome sequences from unrelated strains (J99 and 26,695) has allowed an analysis of the level of diversity from a large-scale yet detailed perspective. Although the two chromosomes are organized differently in a limited number of discrete regions, the genome **size** and gene order of these two "high-virulence" (cagA+ and vacA+) H. **pylori** isolates was found to be highly similar. The regions of organizational difference are associated with insertion sequences, DNA restriction/modification genes, repeat sequences, or a combination of the above. A significant level of variation at the nucleotide level is seen across the genome, providing an explanation for why the nucleotide-based typing techniques have such high discriminatory power among independent H. **pylori** isolates. This nucleotide variation together with the organizational rearrangements appears to have provided an over-estimation of the gene order diversity of H. **pylori** as assessed by pulse-field gel **electrophoresis** . Functional assignments are assigned to approximately only 60% of the gene products in each strain, with one-half of the remaining gene products of unknown function having homologues in other bacteria, while the remainder appear to be H. **pylori** -specific. Between 6% and 7% of the coding capacity of each strain are genes that are absent from the other strain, with almost one-half of these strain-specific genes located in a single hypervariable region called the plasticity zone. The majority of the strain-specific genes in each strain are also H. **pylori** -specific, with no homologues being identified in the public databases. Significantly, over one-half of the functionally assigned strain-specific genes in both H. **pylori** J99 and **26695** encode DNA restriction/modification enzymes. Analysis of the level of conservation between orthologues from the two strains indicates that the H. **pylori** specific genes have a lower level of conservation than those orthologues to which a putative function can be assigned. The plasticity zone represents one of several regions across each genome that is comprised of lower (G+C)% content DNA, some of which has been detected in self-replicating plasmids, suggesting that both horizontal transfer from other species and plasmid integration are responsible for the strain-specific diversity at this locus. These analyses have yielded results with important implications for understanding the genetic diversity of H. **pylori** and its associated diseases, and imply a need to reassess the respective roles of bacterial and host factors in H. **pylori** associated diseases. (80 Refs.)

Tags: Human
Descriptors: Genome, Bacterial; * **Helicobacter pylori** --genetics--GE; *Variation (Genetics); Genes, Bacterial; **Helicobacter** Infections --genetics--GE; **Helicobacter** Infections--microbiology--MI; Phenotype
Record Date Created: 20000310
Record Date Completed: 20000310

1/9/24

10451426 PMID: 10547695

Contributions of genome sequencing to understanding the biology of Helicobacter pylori .

Ge Z; Taylor D E
Division of Comparative Medicine, Massachusetts Institute of Technology, Cambridge 02139, USA.

Annual review of microbiology (UNITED STATES) 1999, 53 p353-87, ISSN 0066-4227 Journal Code: 0372370
Document type: Journal Article; Review; Review, Academic
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

About half of the world's population carries **Helicobacter pylori** , a gram-negative, spiral bacterium that colonizes the human stomach. The link between H. **pylori** and, ulceration as well as its association with the development of both gastric cancer and mucosa-associated lymphoid tissue lymphoma in humans is a serious public health concern. The publication of the genome sequences of two stains of H. **pylori** gives rise to direct evidence on the genetic diversity reported previously with respect to gene organization and nucleotide variability from strain to strain. The genome **size** of H. **pylori** strain **26695** is 1,6697,867 bp and is 1,643,831 bp for strain J99. Approximately 89% of the predicted open reading frames are common to both of the strains, confirming H. **pylori** as a single species. A region containing approximately 45% of H. **pylori** strain-specific open reading frames, termed the plasticity zone, is present on the chromosomes, verifying that some strain variability exists. Frequent alteration of nucleotides in the third position of the triplet codons and various copies of insertion elements on the individual chromosomes appear to contribute to distinct polymorphic fingerprints among strains analyzed by restriction fragment length polymorphisms, random amplified polymorphic DNA method, and repetitive element-polymerase chain reaction. Disordered chromosomal locations of some genes seen by pulsed-field gel **electrophoresis** are likely caused by rearrangement or inversion of certain segments in the genomes. Cloning and functional characterization of the genes involved in acidic survival, vacuolating toxin, cag-pathogenicity island, motility, attachment to epithelial cells, natural transformation, and the biosynthesis of lipopolysaccharides have considerably increased our understanding of the molecular genetic basis for the pathogenesis of H. **pylori** . The homopolymeric nucleotide tracts and dinucleotide repeats, which potentially regulate the on- and off-status of the target genes by the strand-slipped mispairing mechanism, are often found in the genes encoding the outer-membrane proteins, in enzymes for lipopolysaccharide synthesis, and within DNA modification/restriction systems. Therefore, these genes may be involved in the H. **pylori** -host interaction. (178 Refs.)

Tags: Human; Support, Non-U.S. Gov't
Descriptors: Genome, Bacterial; * **Helicobacter pylori** --genetics--GE; * **Helicobacter pylori** --physiology--PH; Amino Acid Sequence; Base Sequence; Evolution, Molecular; **Helicobacter pylori** --pathogenicity--PY; Molecular Sequence Data; Sequence Analysis, DNA; Virulence--genetics--GE
Record Date Created: 19991206
Record Date Completed: 19991206

1/9/25
DIALOG(R)File 155:MEDLINE(R)

09370220 PMID: 1629897
Motility as a factor in the colonisation of gnotobiotic piglets by Helicobacter pylori .
Eaton K A; Morgan D R; Krakowka S
Department of Veterinary Pathobiology, College of Veterinary Medicine, Ohio State University, Columbus 43210.
Journal of medical microbiology (ENGLAND) Aug 1992, 37 (2) p123-7, ISSN 0022-2615 Journal Code: 0224131
Contract/Grant No.: AI07938-02; AI; NIAID; DK39570-01A3; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Non-motile variants of **Helicobacter pylori** (strain **26695**) occurred with a frequency of 1.6 (SD 0.4) x 10(-4) variants/cell/division cycle, and reversion to the motile form occurred with a frequency of less than 10(-7) variants/cell/division cycle. The two forms remained greater than 90% pure for up to 50 cell divisions and differed only in the presence or absence of motility and flagella. Bacteria were recovered from nine of 10 gnotobiotic piglets inoculated orally with motile H. **pylori** , but from only two of eight inoculated with the non-motile variant. The motile form survived for 21 days in infected piglets, but the non-motile variant survived for only 6 days. Bacteria recovered from piglets inoculated with the non-motile variant were non-motile. These data support the hypothesis that motility is a colonisation factor for H. **pylori** .

Tags: Human; Support, U.S. Gov't, P.H.S.

Descriptors: Flagella--physiology--PH; * **Helicobacter pylori** --pathogenicity--PY; Animals; Cell Movement; Colony Count, Microbial; **Electrophoresis** , Polyacrylamide Gel; Germ-Free Life; Swine; Variation (Genetics)

Record Date Created: 19920820

Record Date Completed: 19920820

```
?s 30 (5n) (kda or kd or da or dalton? or kilodalt? or rmw or mw?)
          577451  30
           94490  KDA
           45733  KD
           57221  DA
           12966  DALTON?
            6702  KILODALT?
              25  RMW
           10391  MW?
      S2    7210  30 (5N) (KDA OR KD OR DA OR DALTON? OR KILODALT? OR RMW
                  OR MW?)
?s s2 and (pylori? or helicobact?)
            7210  S2
           29544  PYLORI?
           19051  HELICOBACT?
      S3      47  S2 AND (PYLORI? OR HELICOBACT?)
?s s3 and 26695
              47  S3
             116  26695
      S4       0  S3 AND 26695
?t s3/9/all
```

3/9/1

DIALOG(R)File 155:MEDLINE(R)

16214422 PMID: 15156908

Serological responses of FldA and small-molecular-weight proteins of Helicobacter pylori **: correlation with the presence of the gastric MALT tissue.**

Yang Hsiao-Bai; Sheu Bor-Shyang; Wang Jin-Town; Lin Shih-Ting; Wu Jiunn-Jong

Department of Pathology, National Cheng Kung University, Medical College, Tainan, Taiwan.

Helicobacter (United States) Feb 2004, 9 (1) p81-6, ISSN 1083-4389 Journal Code: 9605411

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: In Process

Subfile: INDEX MEDICUS

PURPOSE: We tested whether the serological response to Flavodoxin-A (FldA) protein and anti- **Helicobacter pylori** immunoblots correlated to the degree of mucosaassociated lymphoid tissue (MALT) in the stomach. METHODS: Eighty H. pyloni-infected patients with different degrees of MALT in the stomach were investigated with serum sampling and endoscopy on enrolment, the 2nd and the 12th months after anti-H. **pylori** therapy. All sera were tested for the anti-FldA protein and anti-H. pylon immunoblots,

including 19.5, 26.5, **30** , 35, 89, and 116 **KDa** (CagA). Regression of follicular gastritis was assessed by histology. RESULTS: Patients with dense lymphoid follicles had higher prevalence rates of anti-FldA protein, 19.5, 26.5, and **30** **KDa** antibodies of H. **pylori** ($p < .05$). Histologic downgrade of MALT was observed in 25% (10/40) of patients in the 2nd month and in 60% (23/38) in the 12th month after H. **pylori** therapy. After H. **pylori** eradication, the patients with MALT and dense lymphoid follicles had significantly negative seroconversions of 19.5, 26.5, **30** , and 35 **KDa** antibodies ($p < .05$), but not of CagA and FldA. CONCLUSION: Patients with gastric MALT and dense lymphoid follicles had different anti-H. **pylori** serological responses to those with scanty or an absence of lymphoid follicles. The negative seroconversion of the smaller-molecular-weight proteins, but not CagA and FldA, may correlate with the regression of MALT by H. **pylori** eradication.

Record Date Created: 20040525

3/9/2

DIALOG(R)File 155:MEDLINE(R)

15742100 PMID: 12557344

[Construction of hpaA gene-engineered attenuated Salmonella typhimurium vaccine strain]

Zhu Senlin; Chen Minhu; Chen Jie; Hu Pinjin; Li Guoqing

Department of Gastroenterology, First Affiliated Hospital, Sun Yat-Sen University of Medical Sciences, Guangzhou 510080, China.

Wei sheng wu xue bao = Acta microbiologica Sinica (China) Feb 2002, 42 (1) p27-32, ISSN 0001-6209 Journal Code: 21610860R

Document type: Journal Article ; English Abstract

Languages: CHINESE

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

To express **Helicobacter** **pylori** hpaA gene in attenuated Salmonella typhimurium vaccine vehicle, and elucidate the potential value of attenuated Salmonella typhimurium as a vector expressing **Helicobacter** **pylori** antigens, by means of molecular biology, 783 bp hpaA gene was cloned into NcoI-SalI site of a procaryotic expression plasmid pTrc99A, and the recombinant plasmid was then used to transform an attenuated Salmonella typhimurium vaccine strain SL3261, and the positive clones were screened by PCR and restriction enzyme digestion. HpaA expression was analyzed by SDS-PAGE and Western blot. Two and 10 days after recombinant strain intragastric immunization, the C57BL/6 mice was sacrificed, and the spleen and terminal ileum was cultured for recombinant strain. The results showed that a recombinant procaryotic expression plasmid pTrc99A-hpaA was constructed, and the recombinant plasmid was then introduced into an attenuated Salmonella typhimurium vaccine strain SL3261 successfully. HpaA was expressed in the recombinant strains as a **30** **kD** protein, and also its immunogenicity was confirmed by Western blot. Recombinant strain was found in both spleen and terminal ileum of each mouse two and ten days after intragastric immunization. We concluded that a recombinant live attenuated Salmonella typhimurium vaccine strain expressing **Helicobacter** **pylori** hpaA gene was constructed and identified, and this work will help to develop oral recombinant live vaccine strains against **Helicobacter** **pylori** infection.

Tags: Support, Non-U.S. Gov't

Descriptors: Genes, Bacterial; * **Helicobacter** **pylori** --genetics--GE; *Hemagglutinins--genetics--GE; *Lipoproteins--genetics--GE; *Salmonella typhimurium--genetics--GE; Amino Acid Sequence; Animals; Bacterial Proteins --biosynthesis--BI; Bacterial Proteins--genetics--GE; Bacterial Vaccines; Base Sequence; **Helicobacter** **pylori** --isolation and purification--IP; Hemagglutinins--biosynthesis--BI; Ileum--microbiology--MI; Lipoproteins --biosynthesis--BI; Mice; Mice, Inbred C57BL; Molecular Sequence Data; Plasmids; Salmonella typhimurium--metabolism--ME; Spleen--microbiology--MI; Transformation, Genetic; Vaccines, Attenuated

CAS Registry No.: 0 (Bacterial Proteins); 0 (Bacterial Vaccines); 0

(Hemagglutinins); 0 (HpaA protein); 0 (Lipoproteins); 0 (Plasmids); 0 (Vaccines, Attenuated)
Record Date Created: 20030131
Record Date Completed: 20040302

3/9/3
DIALOG(R)File 155:MEDLINE(R)

15660699 PMID: 14532338
Increased prevalence of seropositivity for non-gastric Helicobacter **species in patients with autoimmune liver disease.**
Nilsson Ingrid; Kornilovs'ka Iryna; Lindgren Stefan; Ljungh Asa; Wadstrom Torkel
Department of Medical Microbiology, Dermatology and Infection, Division of Bacteriology, Lund University, Solvegatan 23, S-223 62 Lund, Sweden. ingrid.nilsson@mmb.lu.se
Journal of medical microbiology (England) Nov 2003, 52 (Pt 11) p949-53, ISSN 0022-2615 Journal Code: 0224131
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Various **Helicobacter** species have been isolated from the stomach, intestinal tract and liver of a variety of mammalian and some avian species, and **Helicobacter** DNA has been detected in human bile and liver samples. An immunoblot assay was established to analyse serum antibody responses to non-gastric **Helicobacter** species in patients with autoimmune liver diseases, in comparison with healthy individuals. Sera from 36 patients with primary sclerosing cholangitis (PSC), 21 with primary biliary cirrhosis, 19 with autoimmune chronic hepatitis and 80 blood donors were analysed by immunoblot, using cell-surface proteins from **Helicobacter** pullorum, **Helicobacter** bilis and **Helicobacter** hepaticus as antigens. Prior to testing, sera were cross-absorbed with a whole-cell lysate of **Helicobacter pylori** . Antibody reactivity to various proteins of these three **Helicobacter** species was measured by densitometric scanning and results were processed by computer software to estimate antigenic specificity. Results were also compared with antibody response to H. **pylori** . For H. pullorum, reactivity to at least two of the proteins with molecular masses of 48, 45, 37, 20 and 16 kDa, for H. hepaticus, reactivity to the 76, **30** and 21 **kDa** proteins and for H. bilis, reactivity to the 22 and 20 kDa proteins, seemed to have high specificity. Positive immunoblot results with sera from patients with PSC to antigens of H. pullorum, H. bilis and H. hepaticus were found in 38, 22 and 25 % of cases, respectively, and from patients with other autoimmune liver diseases, in 30, 22 and 22 % of cases, respectively. Prevalence of serum antibodies to non-gastric **Helicobacter** species was significantly higher in patients with autoimmune chronic liver diseases than in healthy blood donors ($P < 0.001$). Increased antibody levels to enterohepatic **Helicobacter** species raise questions concerning an infectious role of these emerging bacterial pathogens in human autoimmune liver diseases.
Tags: Human; Support, Non-U.S. Gov't
Descriptors: Antibodies, Bacterial--blood--BL; *Autoimmune Diseases --microbiology--MI; * **Helicobacter** --isolation and purification--IP; *Liver Diseases--microbiology--MI; Adult; Aged; Aged, 80 and over; **Helicobacter** --immunology--IM; Middle Aged; Seroepidemiologic Studies
CAS Registry No.: 0 (Antibodies, Bacterial)
Record Date Created: 20031008
Record Date Completed: 20040217

3/9/4
DIALOG(R)File 155:MEDLINE(R)

14472486 PMID: 10469189

Different penicillin-binding protein profiles in amoxicillin-resistant Helicobacter pylori .

Dore M P; Graham D Y; Sepulveda A R

Department of Medicine and Pathology, VA Medical Center and Baylor College of Medicine, Houston, TX 77030, USA.

Helicobacter (UNITED STATES) Sep 1999, 4 (3) p154-61, ISSN 1083-4389 Journal Code: 9605411

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

BACKGROUND: The beta-lactam group of antibiotics kills bacteria by inhibiting the terminal stages of peptidoglycan metabolism. We have recently identified amoxicillin-resistant **Helicobacter pylori** , none of which expressed beta-lactamase. Penicillin-binding proteins (PBPs) represent a group of target enzymes for the beta-lactam antibiotic family, and alterations in PBPs have been described in other penicillin-resistant bacteria. The amoxicillin-resistant phenotype characteristically was lost after freezing but could be restored by consecutive transfers into gradient plates. MATERIALS AND METHODS: To determine whether amoxicillin resistance in H. **pylori** was related to alterations in any of the H. **pylori** PBPs, five H. **pylori** strains resistant to amoxicillin and three amoxicillin-sensitive strains were tested. PBPs were extracted from bacteria grown to logarithmic phase, labeled in vivo with 3H-benzylpenicillin, and analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) and fluorography. Four main PBPs were separated from all amoxicillin-sensitive H. **pylori** strains. RESULTS: Only three of the four main PBPs were found in the amoxicillin-resistant H. **pylori** strains. The differentially detectable PBP (PBP D) had an apparent molecular weight of **30** to 32 **kD** . CONCLUSION: These results suggest that PBP D might play a role in the amoxicillin-resistant phenotype of H. **pylori** strains lacking beta-lactamase activity.

Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.

Descriptors: Amoxicillin--pharmacology--PD; *Ampicillin Resistance; *Bacterial Proteins; *Carrier Proteins--analysis--AN; * **Helicobacter pylori** --chemistry--CH; * **Helicobacter pylori** --drug effects--DE; *Hexosyltransferases; *Muramoylpentapeptide Carboxypeptidase--analysis--AN; *Peptidyltransferase; Dyspepsia--microbiology--MI; Electrophoresis, Polyacrylamide Gel; **Helicobacter** Infections--microbiology--MI; **Helicobacter pylori** --enzymology--EN; **Helicobacter pylori** --growth and development--GD; Microbial Sensitivity Tests; Penicillins--pharmacology --PD; Peptic Ulcer--microbiology--MI; beta-Lactamases--metabolism--ME

CAS Registry No.: 0 (Bacterial Proteins); 0 (Carrier Proteins); 0 (Penicillins); 26787-78-0 (Amoxicillin)

Enzyme No.: EC 2.3.2.12 (Peptidyltransferase); EC 2.4.1.- (Hexosyltransferases); EC 3.4.16.4 (penicillin-binding protein); EC 3.4.17.8 (Muramoylpentapeptide Carboxypeptidase); EC 3.5.2.6 (beta-Lactamases)

Record Date Created: 19991020

Record Date Completed: 19991020

3/9/5

DIALOG(R)File 155:MEDLINE(R)

14447175 PMID: 10445790

Seroprevalence of eight Helicobacter pylori **antigens among 182 patients with peptic ulcer, MALT gastric lymphoma or non-ulcer dyspepsia. Higher rate of seroreactivity against CagA and 35-kDa antigens in patients with peptic ulcer originating from Europe and Africa.**

Lamarque D; Gilbert T; Roudot-Thoraval F; Deforges L; Chaumette M T; Delchier J C

Service d'Hepatologie et de Gastroenterologie, Centre Hospitalier Universitaire Henri Mondor, Creteil, France. lamarque@im3.inserm.fr

European journal of gastroenterology & hepatology (ENGLAND) Jul 1999, 11 (7) p721-6, ISSN 0954-691X Journal Code: 9000874
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

BACKGROUND: It has been suggested that **Helicobacter pylori** may induce more or less severe gastroduodenal disease according to the strain virulence. DESIGN: We used Western blot to determine antigenic profiles associated with duodenal or gastric ulcer disease, MALT lymphoma and non-ulcer dyspepsia, and to identify geographical differences. METHODS: One hundred and eighty-two consecutive patients with H. **pylori** infection were studied. H. **pylori** infection was diagnosed by a rapid urease test or histological examination of gastric biopsy samples. Bacterial density and gastritis were assessed histologically by using the Sydney scoring system. Western blot was used to identify antibodies against eight antigens (CagA, VacA, urease A, heat shock protein B, and 19.5, 26.5, **30** and 35 **kDa**). Patients were questioned on their smoking habits and place of birth and childhood. RESULTS: There were 73 patients with duodenal ulcer, 30 with gastric ulcer, eight with erosive duodenitis, 17 with gastric MALT lymphoma and 54 with non-ulcer dyspepsia. Most (>85%) were seropositive for the heat shock protein B and 26.5-kDa antigens. The prevalence of the other antigens ranged from 45% (VacA) to 68% (urease B). The seroprevalence of CagA antigen was significantly higher ($P < 0.01$) in cases of gastroduodenal ulcer (84%) than non-ulcer dyspepsia (37%). Similarly, 35-kDa antigen reactivity was more frequent ($P < 0.05$) in duodenal ulcer patients (75%) than in those with non-ulcer dyspepsia (50%). The antigenic profiles associated with MALT gastric lymphoma and non-ulcer dyspepsia were similar. Multivariate analysis showed that only gastroduodenal ulcer was significantly associated with CagA. Gastroduodenal ulcer and a childhood spent in Africa were both associated with 35-kDa and combined CagA-35-kDa reactivity. CONCLUSIONS: This study confirms the strong seroprevalence of H. **pylori** CagA antigen and shows a high prevalence of the 35-kDa antigen in patients with gastroduodenal ulcer, especially those raised in Africa. There was no difference in the serological pattern between patients with non-ulcer dyspepsia and those with MALT lymphoma. Tests for antibodies to the CagA-35-kDa antigen combination might be used to select H. **pylori** -infected dyspeptic patients warranting treatment.

Tags: Human
Descriptors: Antigens, Bacterial--blood--BL; *Dyspepsia--immunology--IM; * **Helicobacter** Infections--immunology--IM; * **Helicobacter pylori** --immunology--IM; *Lymphoma, Mucosa-Associated Lymphoid Tissue--immunology --IM; *Peptic Ulcer--immunology--IM; *Stomach Neoplasms--immunology--IM; Africa--epidemiology--EP; Aldehyde Reductase; Antibodies, Bacterial --analysis--AN; Bacterial Proteins--immunology--IM; Blotting, Western; Duodenal Ulcer--epidemiology--EP; Duodenal Ulcer--immunology--IM; Duodenal Ulcer--microbiology--MI; Dyspepsia--microbiology--MI; Europe --epidemiology--EP; Genes, Bacterial; Heat-Shock Proteins--immunology--IM; **Helicobacter** Infections--epidemiology--EP; Lymphoma, Mucosa-Associated Lymphoid Tissue--microbiology--MI; Peptic Ulcer--epidemiology--EP; Peptic Ulcer--microbiology--MI; Seroepidemiologic Studies; Statistics, Nonparametric; Stomach Neoplasms--microbiology--MI; Stomach Ulcer --epidemiology--EP; Stomach Ulcer--immunology--IM; Stomach Ulcer --microbiology--MI
CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (Heat-Shock Proteins); 0 (cagA protein, Helicobacter pylori); 0 (hypertonic stress protein, 35-kDa)
Enzyme No.: EC 1.1.1.21 (Aldehyde Reductase)
Record Date Created: 19990908
Record Date Completed: 19990908

3/9/6

14301196 PMID: 10203471

Serological discrimination of dogs infected with gastric Helicobacter **spp. and uninfected dogs.**

Strauss-Ayali D; Simpson K W; Schein A H; McDonough P L; Jacobson R H; Valentine B A; Peacock J

Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, New York 14853, USA.

Journal of clinical microbiology (UNITED STATES) May 1999, 37 (5) p1280-7, ISSN 0095-1137 Journal Code: 7505564

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Characterization of the humoral immune responses of people to **Helicobacter pylori** infection has facilitated the investigation of the host response to bacterial virulence factors and the development of sensitive and specific diagnostic tests. Dogs are commonly infected with gastric **Helicobacter** spp., but the presence of multiple **Helicobacter** spp. and possible coinfection in individual dogs have complicated serological evaluation. Evaluation of the antigenic homology of **Helicobacter** spp. revealed that the major protein bands of **Helicobacter** felis and **Helicobacter** bizzozeronii, two **Helicobacter** spp. that infect dogs, were very similar to UreA (29 to 31 kDa), UreB (63 to 66 kDa), and HSP (58 to 60 kDa) of H. **pylori** , and sera from infected and uninfected dogs bound in a similar way to each antigen. Immunoblotting and an enzyme-linked immunosorbent assay (ELISA) with H. felis ATCC 49179 antigen were performed with 101 serum samples (from 78 infected dogs and 23 uninfected dogs). Samples from uninfected dogs (median = 8) had fewer bands on immunoblotting than samples from infected dogs (median = 16) ($P < 0.05$). Combinations of the presence of any two of the low-molecular-mass bands (19, 25, **30** , 32, and 37 **kDa**) or the high-molecular-mass bands (86 and 94 kDa) were found almost solely in samples from infected dogs ($P < 0.0001$). Kinetic ELISA results were significantly higher for samples from infected dogs (median = 0. 0802 optical density unit [OD]/min) than for samples from uninfected dogs (median = 0.01428 OD/min). The combination of ELISA and immunoblotting results gave a specificity of 95.6% and a sensitivity of 79.8%. No correlation between ELISA results, colonization density, degree of inflammation, and presence of lymphoid follicles was observed. The results indicate substantial antigenic homology between H. felis, H. **pylori** , and H. bizzozeronii. The combination of ELISA and immunoblotting was a highly specific and moderately sensitive indicator of infection. The degree of seropositivity assessed by ELISA was not related to bacterial colonization density, the degree of gastric inflammation, or the presence of lymphoid follicles.

Tags: Female; Support, Non-U.S. Gov't

Descriptors: **Helicobacter** Infections--immunology--IM; * **Helicobacter pylori** --immunology--IM; *Stomach--microbiology--MI; Animals; Antigens, Bacterial--analysis--AN; Dogs; Enzyme-Linked Immunosorbent Assay; Gastritis --etiology--ET; **Helicobacter** Infections--diagnosis--DI; Immunoblotting; Immunoglobulin G--blood--BL; Molecular Weight; Serologic Tests

CAS Registry No.: 0 (Antigens, Bacterial); 0 (Immunoglobulin G)

Record Date Created: 19990507

Record Date Completed: 19990507

3/9/7

14220012 PMID: 9952387

Qualitative and quantitative analysis of the local and systemic antibody response in mice and humans with Helicobacter **immunity and infection.**

Blanchard T G; Nedrud J G; Reardon E S; Czinn S J

Department of Pediatrics, Case Western Reserve University, Cleveland, OH 44106, USA. tgb4@po.cwru.edu

Journal of infectious diseases (UNITED STATES) Mar 1999, 179 (3)

p725-8, ISSN 0022-1899 Journal Code: 0413675
Contract/Grant No.: AI-40701; AI; NIAID; DK-46461; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: AIM; INDEX MEDICUS
Immunization can prevent or cure an otherwise chronic gastric **Helicobacter** infection in several different animal models. The goal of the present study was to compare the titers and specificities of local and systemic antibody responses generated by **Helicobacter** infection and immunization. Protective immunization results in levels of specific gastric antibody significantly lower than induced by infection. However, antibodies from protectively immunized mice preferentially recognize immunodominant proteins of 10-22 and **30 kDa** . Immunoblot analysis of infected mice and humans demonstrated that the serum IgA, but not serum IgG, binding profiles yield an accurate profile of the antigenic specificity of the host's gastric IgA. Therefore, serum IgA may be useful in evaluating the immunodominant antigens at the gastric mucosa of infected persons and possibly in determining the immunogenicity of orally applied **Helicobacter** vaccines.
Tags: Female; Human; Male; Support, U.S. Gov't, P.H.S.
Descriptors: Bacterial Vaccines; * **Helicobacter** --immunology--IM; * **Helicobacter** Infections--immunology--IM; * **Helicobacter pylori** ; Adult; Aged; Animals; Antibodies, Bacterial--immunology--IM; Antibody Formation; Antigens, Bacterial--immunology--IM; Dyspepsia--immunology--IM; Dyspepsia --microbiology--MI; Gastric Mucosa--immunology--IM; **Helicobacter pylori** --immunology--IM; Immunoglobulin A--immunology--IM; Mice; Mice, Inbred C57BL; Middle Aged; Peptic Ulcer--immunology--IM; Peptic Ulcer --microbiology--MI
CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Vaccines); 0 (Immunoglobulin A)
Record Date Created: 19990330
Record Date Completed: 19990330

3/9/8
DIALOG(R)File 155:MEDLINE(R)

14188581 PMID: 9890472
The prevalence of antibody to CagA in children is not a marker for specific disease.
Mitchell H M; Hazell S L; Bohane T D; Hu P; Chen M; Li Y Y
The School of Microbiology and Immunology, University of New South Wales, Sydney, Australia.
Journal of pediatric gastroenterology and nutrition (UNITED STATES) Jan 1999, 28 (1) p71-5, ISSN 0277-2116 Journal Code: 8211545
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
BACKGROUND: In adults, a high prevalence of antibody to the cytotoxin-associated antigen (CagA) of **Helicobacter pylori** has been linked to the development of more serious gastroduodenal disease. Few investigators have examined this association in children. The purpose of this study was to investigate the seroprevalence of antibody to the CagA antigen as well as other specific H. **pylori** antigens in children. METHODS: By use of an immunoblot analysis kit, the immune response to specific H. **pylori** antigens in serum collected from 21 H. **pylori** -positive symptomatic Australian children, 5 with peptic ulcer disease and 16 with nonulcer dyspepsia, and 33 H. **pylori** -positive asymptomatic Chinese children. Sera from 20 H. **pylori** -negative symptomatic Australian children were used as control subjects. RESULTS: Antibody responses to the 26.5- **kDa** , **30** - **kDa** , and 116- **kDa** (CagA) antigens were found to be the most prevalent, with 81.5%, 79.6%, and 76% of children, respectively,

mounting a response. In contrast, antibody responses to the 19.5-kDa, 35-kDa, 45-kDa, 60-kDa, 89 kDa (VacA), and 180-kDa antigens occurred in 55.5%, 24%, 16.7%, 63%, 37%, and 7.4% of children, respectively. A higher prevalence of antibody response to CagA was found in the symptomatic Australian children with peptic ulcer disease (100%) compared with prevalence in those with nonulcer dyspepsia (56.3%), but the difference did not reach statistical significance. No significant difference was found between the prevalence of antibody to CagA in the Australian peptic ulcer disease group (100%) and that in the asymptomatic Chinese children (81.8%). CONCLUSION: These results suggest that in children CagA is not a marker of specific disease development.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: Antibodies, Bacterial--blood--BL; *Antigens, Bacterial --immunology--IM; *Bacterial Proteins--immunology--IM; *Gastrointestinal Diseases--microbiology--MI; * **Helicobacter** Infections--immunology--IM; * **Helicobacter pylori** --immunology--IM; Adolescent; Child; Child, Preschool; Gastrointestinal Diseases--pathology--PA; **Helicobacter** Infections--epidemiology--EP; **Helicobacter** Infections--pathology--PA; Immunoblotting; Infant; Seroepidemiologic Studies

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (cagA protein, Helicobacter pylori)

Record Date Created: 19990323

Record Date Completed: 19990323

3/9/9

DIALOG(R)File 155:MEDLINE(R)

14063759 PMID: 10607420

Evaluation of a western blot technique (Helicoblot 2.0) fpr the detection of specific Heliocobacter pylori **antigens in children)]**

Evaluacion de una tecnica de western-blot (Helicoblot 2.0) para la deteccion de anticuerpos frente a antigenos especificos de **Helicobacter pylori** en ninos.

Lopez-Brea M; Alarcon T; Domingo D; Sanchez I; Martinez M J; Sanz J C

Servicio de Microbiologia, Hospital Universitario de la Princesa, Madrid.

Enfermedades infecciosas y microbiologia clinica (SPAIN) Jun-Jul 1998, 16 (6) p275-9, ISSN 0213-005X Journal Code: 9104081

Document type: Journal Article ; English Abstract

Languages: SPANISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

BACKGROUND: The aim of this study was to evaluate the diagnosis usefulness of antibodies detection against different specific antigens of H. **pylori** (Hp) in children by a Western-blot commercially available (Helicoblot 2.0). MATERIAL AND METHODS: 96 symptomatic paediatric patients in two groups were studied. Group 1: 48 patients Hp positive by culture and/or histology (mean age, 10.19 +/- 3.40 years; 27 boys and 21 girls) and group 2: 48 patients Hp negative by both culture and histology (mean age, 9.31 +/- 3.15 years; 34 boys and 14 girls). Qualitative IgG detection against the protein antigens of 19.5 kD (urease E), 26.5 **kD** (urease D), **30 kD** (urease H), 35 **kD** , 89 kD (vacuolating toxin VacA) and 116 kD (cagA protein) was determined by a Western-blot assay (Helicoblot 2.0, Genelabs, Singapore). RESULTS: A specificity of 100% was obtained when reactivity against 19.5 kD protein (urease E) was considered alone. However, the best accuracy levels were observed with 26.5 **kD** (urease A) and **30 kD** (urease H) antigens. An accuracy of 88.5% was observed when the manufacturer's recommended criteria was used (positivity of at least 116, 89 or 35 kD single bands or positivity of at least 2 bands of the **30** , 26.5 or 19.5 **kD**). CONCLUSIONS: Helicoblot 2.0 is a good method to diagnosis Hp infection in children. A criteria combining IgG against 2 urease antigens (preferentially urease A) or one urease antigen and a virulence associated protein (CagA o VacA) is recommended.

Tags: Female; Human; Male

Descriptors: Antibodies, Bacterial--blood--BL; *Antigens, Bacterial

--immunology--IM; *Blotting, Western--methods--MT; * **Helicobacter** Infections--diagnosis--DI; * **Helicobacter pylori** --immunology--IM; Adult ; Bacterial Proteins--immunology--IM; Child; Evaluation Studies; Gastroscopy; **Helicobacter** Infections--microbiology--MI; Reagent Kits, Diagnostic; Urease--immunology--IM
CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (Reagent Kits, Diagnostic); 0 (cagA protein, Helicobacter pylori); 0 (vacuolating toxin, Helicobacter pylori)
Enzyme No.: EC 3.5.1.5 (Urease)
Record Date Created: 19990125
Record Date Completed: 19990125

3/9/10
DIALOG(R)File 155:MEDLINE(R)

13961513 PMID: 9660771
Affinity purification of Helicobacter pylori **urease. Relevance to gastric mucin adherence by urease protein.**
Icatlo F C; Kuroki M; Kobayashi C; Yokoyama H; Ikemori Y; Hashi T; Kodama Y
Immunology Research Institute, Ghen Corporation, 839-1 Sano, Gifu City 501-11, Japan.
Journal of biological chemistry (UNITED STATES) Jul 17 1998, 273 (29) p18130-8, ISSN 0021-9258 Journal Code: 2985121R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
A simple, reproducible and high yield method of **Helicobacter pylori** urease enzyme purification was developed using a heparinoid (Cellufine sulfate) affinity gel. The purification method involved two sequential steps using the same gel that takes advantage of the differential affinity of urease to the heparinoid at two levels of hydrogen ion concentration. SDS-polyacrylamide gel electrophoresis analysis of affinity-purified urease revealed two major protein bands with about 62- and **30 - kDa** molecular mass. When whole cell lysates of clinical and laboratory strains of H. **pylori** were probed by Western blot, anti-urease hyperimmune serum produced by affinity-purified urease in rabbit recognized only the two bands corresponding to the urease A and B subunits. To probe the molecular relevance of affinity gel adherence to mucin adherence, the purified urease was derivatized with N-hydroxysuccinimidobiotin and used in adherence assays. Competitive inhibition tests revealed commonality of urease receptors among gastric mucin, heparin, and heparinoid. Composite data on adherence kinetics modulated by pH, salt, incubation time, and concentration of urease or mucin were indicative of conformation-dependent ligand-receptor interaction.
Tags: In Vitro
Descriptors: Gastric Mucin--metabolism--ME; * **Helicobacter pylori** --enzymology--EN; *Urease--isolation and purification--IP; Animals; Binding, Competitive; Biotinylation; Blotting, Western; Chromatography, Affinity; Enzyme-Linked Immunosorbent Assay; Hydrogen-Ion Concentration; Protein Binding; Rabbits; Swine; Urease--metabolism--ME
CAS Registry No.: 0 (Gastric Mucin)
Enzyme No.: EC 3.5.1.5 (Urease)
Record Date Created: 19980813
Record Date Completed: 19980813

3/9/11
DIALOG(R)File 155:MEDLINE(R)

13842245 PMID: 9542911
Use of immunoblot assay to define serum antibody patterns associated with

Helicobacter pylori **infection and with H.** pylori **-related ulcers.**
Aucher P; Petit M L; Mannant P R; Pezennec L; Babin P; Fauchere J L
Department of Microbiology (EA 1720), Centre Hospitalier et Universitaire, Poitiers, France.
Journal of clinical microbiology (UNITED STATES) Apr 1998, 36 (4) p931-6, ISSN 0095-1137 Journal Code: 7505564
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Serology has been used worldwide to detect **Helicobacter pylori** infection. Using an immunoblot assay with an antigen from strain ATCC 43579, we sought to determine the antibodies which were good markers of colonization and the antibody patterns associated with ulcers or atrophy. Out of 98 dyspeptic patients, 41 were colonized by H. **pylori** , based on a positive culture or on positive results of both a urease test and direct examination. These 41 patients were seropositive by an enzyme immunoassay, and 12 of them had ulcers and 29 had evidence of atrophy. Fifty-seven of the 98 patients were noncolonized. Twenty-five of the 57 had evidence of gastric atrophy, and 10 were seropositive; 5 of these 10 had ulcers. By Western blot analysis, 12 antibodies were significantly more frequent in sera from colonized patients, and they produced immunoreactive bands at 125, 87, 74, 66, 54, 48, 46, 42, 35, **30** , 16 and 14 **kDa** . The presence of at least one band at 54, 35, or 42 kDa was the best marker of infection (sensitivity, 95%; specificity, 82%). In the group of colonized patients, none of the antibody patterns were correlated to gastric atrophy. Conversely, the presence of a band at 125, 87, or 35 kDa was statistically associated with the presence of an ulcer. The simultaneous presence of bands at 87 and 35 kDa predicted the risk of ulcers with 83% sensitivity and 69% specificity. By using CagA-positive and VacA-positive strains and CagA-negative and VacA-negative isogenic mutants, the antigens corresponding to the bands at 125 and 87 kDa were shown to be CagA and VacA, respectively. On the other hand, the 35-kDa antigen is a novel uncharacterized component of H. **pylori** . These results may help to optimize the composition of antigenic preparations for serologic detection of H. **pylori** colonization. Immunoblot assay would be useful for screening patients at high risk of ulcers.

Tags: Female; Human; Male; Support, Non-U.S. Gov't
Descriptors: Antibodies, Bacterial--blood--BL; * **Helicobacter** Infections --diagnosis--DI; * **Helicobacter pylori** --immunology--IM; *Peptic Ulcer --microbiology--MI; Adolescent; Adult; Aged; Aged, 80 and over; Child; Immunoblotting; Middle Aged
CAS Registry No.: 0 (Antibodies, Bacterial)
Record Date Created: 19980520
Record Date Completed: 19980520

3/9/12
DIALOG(R)File 155:MEDLINE(R)

13331909 PMID: 9003610
Immunoblot assay for serodiagnosis of Helicobacter pylori **infections.**
Nilsson I; Ljungh A; Aleljung P; Wadstrom T
Department of Medical Microbiology, University of Lund, Sweden.
Journal of clinical microbiology (UNITED STATES) Feb 1997, 35 (2) p427-32, ISSN 0095-1137 Journal Code: 7505564
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

An immunoblot assay for the serological diagnosis of **Helicobacter pylori** infection was evaluated. Serum samples from patients whose gastric biopsy specimens were known to be positive or negative for H. **pylori** on culture were used to establish interpretive criteria for the immunoblot

assay. A panel of sera from patients with diseases other than H. **pylori** infection and sera from healthy blood donors were included to validate these criteria. All sera were initially assessed in an enzyme immunoassay (Ge-EIA), based on acid glycine-extracted cell surface proteins of H. **pylori** NCTC 11637. The same antigen extract was used in the immunoblot assay. In addition, the Ge-EIA and the immunoblot assay were compared with a commercially available EIA (Seradyn, Color Vue **Pylori**). Bands of 110/120 kDa and/or two of five low-molecular-mass proteins (26, 29, **30** , 31, and 33 **kDa** , in any combination) showed a strong correlation with the H. **pylori** culture-positive patients (97.5%) compared to the correlation obtained with the EIA results (Ge-EIA, 87.5%; Seradyn EIA, 92.5%), and the antibody responses to these proteins were considered specific reactions. In 37 of 40 serum samples from culture-negative patients and also in sera from patients with other disorders, a moderate antibody reactivity to the medium-size proteins (43 to 66 kDa) was observed, and these were considered not valuable for a specific immunoblot assay. Among sera from culture-positive patients, 39 of 40 serum samples were defined to be immunoblot positive, and from among sera from culture-negative patients, 3 of 40 serum samples were defined to be immunoblot positive. The use of sera from patients with negative cultures for H. **pylori** as negative controls may decrease the sensitivity due to sampling error and false-negative culture results. Immunoblot assay-positive results were detected among 10% of sera from patients with other diseases, whereas they were detected among 42.5% of sera by the Ge-EIA and 47.5% of sera by the Seradyn-EIA. The higher number of EIA-positive sera in this group reflects a possible cross-reactivity (false-positive EIA result). Of the blood donors, representing asymptomatic but possibly colonized subjects, 24% were immunoblot positive. In conclusion, our data indicate that immunoblotting is more sensitive as well as more specific than EIA. Moreover, it permits detection of antibody responses to specific antigens, e.g., the cytotoxin-associated CagA protein, which may have pathological implications.

Tags: Comparative Study; Human; Support, Non-U.S. Gov't

Descriptors: Antibodies, Bacterial--blood--BL; * **Helicobacter** Infections --diagnosis--DI; * **Helicobacter pylori** --immunology--IM; *Immunoblotting ; Adult; Aged; Aged, 80 and over; Animals; Antibodies, Bacterial --immunology--IM; Antibodies, Monoclonal--immunology--IM; Antibody Specificity; Antigens, Bacterial--immunology--IM; Antigens, Surface --immunology--IM; Evaluation Studies; Immunoenzyme Techniques; Middle Aged; Rabbits; Sensitivity and Specificity

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antibodies, Monoclonal); 0 (Antigens, Bacterial); 0 (Antigens, Surface)

Record Date Created: 19970325

Record Date Completed: 19970325

3/9/13

DIALOG(R)File 155:MEDLINE(R)

13124267 PMID: 8792699

Serological response to specific Helicobacter pylori **antigens: antibody against CagA antigen is not predictive of gastric cancer in a developing country.**

Mitchell H M; Hazell S L; Li Y Y; Hu P J

School of Microbiology and Immunology, University of New South Wales, Sydney, Australia.

American journal of gastroenterology (UNITED STATES) Sep 1996, 91 (9) p1785-8, ISSN 0002-9270 Journal Code: 0421030

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

OBJECTIVES: In symptomatic patients resident in developed countries, a high prevalence of antibody to the cytotoxin-associated antigen (CagA) of **Helicobacter pylori** has been linked to the development of peptic ulcer

disease and gastric cancer. This association has not been examined in developing countries, nor in asymptomatic subjects resident in either developed or developing countries. The aim of this study was to examine the seroprevalence of antibody to the CagA antigen; as well as other specific H. **pylori** antigens in symptomatic and asymptomatic individuals resident in Australia and China. METHODS: The Helico-blot 2.0 Western blot system was used for the detection of antibodies to specific antigens of H. **pylori** in sera obtained from the following H. **pylori** -positive groups: 19 Australian blood donors, 96 Australian nonulcer dyspepsia patients, 29 Australian duodenal ulcer patients, 35 asymptomatic Chinese subjects, and 48 Chinese gastric cancer patients. RESULTS: Nine antigens were commonly recognized by sera from Australian and Chinese subjects. These antigens were of molecular mass 19.5 kDa, 26.5 kDa, 35 kDa, 45 kDa, 60 kDa, 89 kDa (VacA), 116 kDa (CagA), and 180 kDA. A significant association between the prevalence of antibody to the CagA antigen and duodenal ulcer disease was observed in Australian subjects; however, no association between the prevalence of antibody to the CagA antigen and gastric cancer was found in Chinese subjects. In subjects from both countries, a significant association was found between antibody to the **30 - kDa** and 45- **kDa** antigens and more serious gastroduodenal disease. CONCLUSION: The results of this study suggest that the cagA gene is not associated with the development of more serious gastroduodenal disease; however, it cannot be ruled out that this gene may be an important but insufficient factor in some disease processes.

Tags: Comparative Study; Human; Support, Non-U.S. Gov't

Descriptors: Antibodies, Bacterial--blood--BL; *Antigens, Bacterial --immunology--IM; *Bacterial Proteins--immunology--IM; * **Helicobacter** Infections--epidemiology--EP; * **Helicobacter pylori** --immunology--IM; *Stomach Neoplasms--epidemiology--EP; Adult; Aged; Australia--epidemiology --EP; Blotting, Western; China--epidemiology--EP; Developing Countries; Duodenal Ulcer--epidemiology--EP; Duodenal Ulcer--microbiology--MI; Dyspepsia--epidemiology--EP; Dyspepsia--microbiology--MI; Middle Aged; Prevalence; Seroepidemiologic Studies; Stomach Neoplasms--microbiology--MI; Stomach Ulcer--epidemiology--EP; Stomach Ulcer--microbiology--MI

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (cagA protein, Helicobacter pylori)

Record Date Created: 19961017

Record Date Completed: 19961017

3/9/14

DIALOG(R)File 155:MEDLINE(R)

12985137 PMID: 8675345

Changes in Helicobacter pylori **ultrastructure and antigens during conversion from the bacillary to the coccoid form.**

Benaissa M; Babin P; Quellard N; Pezennec L; Cenatiempo Y; Fauchere J L

Laboratoire de Microbiologie A, Centre Hospitalier Regional Universitaire-La Miletrie, Poitiers, France.

Infection and immunity (UNITED STATES) Jun 1996, 64 (6) p2331-5, ISSN 0019-9567 Journal Code: 0246127

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

In vitro, **Helicobacter pylori** converts from a bacillary to a full coccoid form via an intermediate U-shaped form. Organisms with a full coccoid form keep a double membrane system, a polar membrane, and invagination structures. Western blots (immunoblots) of sera from colonized patients show that some high-molecular-mass antigenic fractions are expressed only in coccoids. Conversely, fractions of **30** and 94 **kDa** were more intensively detected in the bacillary forms. These results suggest that (i) coccoid conversion is not a degenerative transformation and (ii) antigens specific to the coccoid forms are expressed in vivo.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: Antigens, Bacterial--analysis--AN; * **Helicobacter pylori** --ultrastructure--UL; Blotting, Western; **Helicobacter pylori** --immunology--IM; Microscopy, Electron
CAS Registry No.: 0 (Antigens, Bacterial)
Record Date Created: 19960812
Record Date Completed: 19960812

3/9/15
DIALOG(R)File 155:MEDLINE(R)

12951522 PMID: 8620891

Catalytic properties, molecular composition and sequence alignments of pyruvate: ferredoxin oxidoreductase from the methanogenic archaeon Methanosarcina barkeri (strain Fusaro).

Bock A K; Kunow J; Glasemacher J; Schonheit P
Institut fur Pflanzenphysiologie und Mikrobiologie, Freien Universitat, Berlin, Germany.

European journal of biochemistry / FEBS (GERMANY) Apr 1 1996, 237 (1) p35-44, ISSN 0014-2956 Journal Code: 0107600
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Methanosarcina barkeri (strain Fusaro) was grown on pyruvate as methanogenic substrate [Bock, A. K., Prieger-Kraft, A. & Schonheit, P. (1994) Arch. Microbiol. 161, 33-46]. The first enzyme of pyruvate catabolism, pyruvate oxidoreductase, which catalyzes oxidation of pyruvate to acetyl-CoA was purified about 90-fold to apparent electrophoretic homogeneity. The purified enzyme catalyzed the CoA-dependent oxidation of pyruvate with ferredoxin as an electron acceptor which defines the enzyme as a pyruvate: ferredoxin oxidoreductase. The deazaflavin, coenzyme F420, which has been proposed to be the physiological electron acceptor of pyruvate oxidoreductase in methanogens, was not reduced by the purified enzyme. In addition to ferredoxin and viologen dyes, flavin nucleotides served as electron acceptors. Pyruvate: ferredoxin oxidoreductase also catalyzed the oxidation of 2-oxobutyrate but not the oxidation of 2-oxoglutarate, indolepyruvate, phenylpyruvate, glyoxylate, 3-hydroxypyruvate and oxaloacetate. The apparent Km values of pyruvate:ferredoxin oxidoreductase were 70 microM for pyruvate, 6 microM for CoA and 30 microM for clostridial ferredoxin. The apparent Vmax with ferredoxin was about 30 U/mg (at 37 degrees C) with a pH optimum of approximately 7. The temperature optimum was approximately 60 degrees C and the Arrhenius activation energy was 40 kJ/mol (between 30 degrees C and 60 degrees C). The enzyme was extremely oxygen sensitive, losing 90% of its activity upon exposure to air for 1 h at 0 degrees C. Sodium nitrite inhibited the enzyme with a Ki of about 10 mM. The native enzyme had an apparent molecular mass of approximately 130 kDa and was composed of four different subunits with apparent molecular masses of 48, **30** , 25, and 15 **kDa** which indicates that the enzyme has an alpha beta gamma delta structure. The enzyme contained 1 mol/mol thiamine diphosphate, and about 12 mol/mol each of non-heme iron and acid-labile sulfur. FAD, FMN and lipoic acid were not found. The N-terminal amino acid sequences of the four subunits were determined. The sequence of the alpha-subunit was similar to the N-terminal amino acid sequence of the alpha-subunit of the heterotetrameric pyruvate:ferredoxin oxidoreductases of the hyperthermophiles Archaeoglobus fulgidus, Pyrococcus furiosus and Thermotoga maritima and of the mesophile **Helicobacter pylori** , and to the N-terminal amino acid sequence of the homodimeric pyruvate:ferredoxin oxidoreductase from proteobacteria and from cyanobacteria. No sequence similarities were found, however, between the alpha-subunit of the M. barkeri enzyme and the heterodimeric pyruvate:ferredoxin oxidoreductase of the archaeon Halobacterium halobium.

Tags: Support, Non-U.S. Gov't
Descriptors: *Ketone Oxidoreductases--genetics--GE; *Ketone Oxidoreductases--metabolism--ME; *Methanosarcina barkeri--enzymology--EN;

Amino Acid Sequence; Catalysis; Cell Extracts; Chromatography, Gel; Chromatography, Ion Exchange; Electrophoresis, Polyacrylamide Gel; Ketone Oxidoreductases--isolation and purification--IP; Molecular Sequence Data; Sequence Homology, Amino Acid
Molecular Sequence Databank No.: GENBANK/X77515; SWISSPROT/P80521; SWISSPROT/P80522; SWISSPROT/P80523; SWISSPROT/P80524
CAS Registry No.: 0 (Cell Extracts)
Enzyme No.: EC 1.2. (Ketone Oxidoreductases); EC 1.2.7.1 (pyruvate synthase)
Record Date Created: 19960619
Record Date Completed: 19960619

3/9/16
DIALOG(R)File 155:MEDLINE(R)

12926908 PMID: 8606074
Antigen recognition during progression from acute to chronic infection with a cagA-positive strain of Helicobacter pylori .
Mitchell H M; Hazell S L; Kolesnikow T; Mitchell J; Frommer D
School of Microbiology and Immunology, The University of New South Wales, Sydney, Australia.
Infection and immunity (UNITED STATES) Apr 1996, 64 (4) p1166-72, ISSN 0019-9567 Journal Code: 0246127
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
We have previously published two reports on acute infection with **Helicobacter pylori** , one of an adult male and one of a family of four. In the present study, we have isolated H. **pylori** from each of twin boys in the family and compared these by use of random amplified polymorphic DNA PCR. In addition, we have monitored the antibody response over time of the family and the adult male by Western blotting (immunoblotting) with two different strains of H. **pylori** as the antigen and by use of a commercial kit. The acutely infected twin boys were infected by an identical strain of H. **pylori** . The twin boys responded to antigens of 19, 26.5, and 29 **kDa** **30** days after the initial diagnosis, with recognition of 43-to 49-, 66-, 69-, and 87-kDa antigens by day 63. One twin responded to the CagA antigen on day 63, whereas the other responded on day 857. Antibody to the CagA antigen was not detected by use of the infecting strain, UNSW-RU1. Investigation of UNSW-RU1 revealed the presence of cagA. In two acutely infected adults (one, the father of the boys), the initial response to a 45-kDA antigen was later followed by responses to 19-, 29-, 49-, 60-, 77-, and 84-kDa antigens. Sera from the twins' younger sister, born 17 months after the twins acute episode, indicated that she also had become infected. This report supports intrafamilial transmission of H. **pylori** . Initial antibody responses in the children were to small-molecular-size antigens; in the adults, the initial responses were to larger-molecular-size antigens. The pattern of the serological response differs according to the antigen used. This has implications in regard to international data comparisons.
Tags: Human; Male; Support, Non-U.S. Gov't
Descriptors: Antigens, Bacterial--immunology--IM; *Bacterial Proteins --analysis--AN; * **Helicobacter** Infections--immunology--IM; * **Helicobacter pylori** --immunology--IM; Acute Disease; Adult; Antibodies, Bacterial --blood--BL; Base Sequence; Blotting, Western; Chronic Disease; DNA Fingerprinting; Immunoglobulin G--blood--BL; Immunoglobulin M--blood--BL; Molecular Sequence Data; Molecular Weight
CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (Immunoglobulin G); 0 (Immunoglobulin M); 0 (cagA protein, Helicobacter pylori)
Record Date Created: 19960523
Record Date Completed: 19960523

boarder. One possible mechanism tested was whether granulocytes are directly activated by water-soluble membrane proteins (WSP) from H. **pylori** . These findings were compared with the effects of WSP from other bacteria (**Helicobacter** felis, Campylobacter jejuni, Escherichia coli, and Staphylococcus aureus). A unique activation pattern by H. **pylori** WSP was found. Like all other WSP tested, they induced an upregulation of CD11b but had no influence on CD11c and, most strikingly, CD62L expression. In contrast, E. coli WSP, e.g., not only induce a strong CD11b and CD11c expression but also lead to a loss in surface CD62L. The lack of CD62L shedding conserves rolling of granulocytes along the endothelium, creating a favorable precondition for granulocytes to stick more readily to activated endothelium after H. **pylori** stimulation via CD11b-CD54 receptor-counterreceptor interaction. This may explain why H. **pylori** infection is a very strong stimulus for granulocyte infiltration. The active fraction for the induction of CD11b on granulocytes is a heat- and protease-sensitive protein with a molecular mass between **30** and 100 **kDa** . One activation step involved may be the binding of WSP to CD15 determinants on granulocytes with subsequent induction of CD11b.

Tags: Human; In Vitro; Support, Non-U.S. Gov't

Descriptors: Bacterial Outer Membrane Proteins--pharmacology--PD; *Cell Adhesion Molecules--metabolism--ME; *Granulocytes--cytology--CY; * **Helicobacter pylori** --pathogenicity--PY; *Integrin alphaXbeta2 --metabolism--ME; *Macrophage-1 Antigen--metabolism--ME; Antigens, Bacterial--immunology--IM; Antigens, CD15--metabolism--ME; Bacterial Proteins--immunology--IM; Bacterial Proteins--pharmacology--PD; Cell Adhesion--drug effects--DE; L-Selectin

CAS Registry No.: 0 (Antigens, Bacterial); 0 (Antigens, CD15); 0 (Bacterial Outer Membrane Proteins); 0 (Bacterial Proteins); 0 (Cell Adhesion Molecules); 0 (Integrin alphaXbeta2); 0 (Macrophage-1 Antigen) ; 126880-86-2 (L-Selectin)

Record Date Created: 19950727

Record Date Completed: 19950727

3/9/19

DIALOG(R)File 155:MEDLINE(R)

12598564 PMID: 7714196

Identification of Helicobacter pylori **by immunological dot blot method based on reaction of a species-specific monoclonal antibody with a surface-exposed protein.**

Bolin I; Lonroth H; Svennerholm A M

Department of Medical Microbiology and Immunology, Goteborg University, Sweden.

Journal of clinical microbiology (UNITED STATES) Feb 1995, 33 (2) p381-4, ISSN 0095-1137 Journal Code: 7505564

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Monoclonal antibodies (MAbs) against membrane preparations of **Helicobacter pylori** were produced. One MAb was found to be specific for H. **pylori** , because it did not react with a number of other bacterial species, including **Helicobacter** felis and Campylobacter jejuni. This MAb reacted with a **30** - **kDa** protein found in outer membrane preparations of H. **pylori** . The protein was also detected on the cell surface on intact bacteria when analyzed by immunoelectron microscopy. To facilitate the identification of H. **pylori** isolates after culturing of biopsies, an immunodot blot assay based on the reaction of this MAb was developed. This assay was found to be highly specific for H. **pylori** . Sixty-six clinical isolates typed as H. **pylori** by conventional biochemical tests were found to be positive, whereas no other bacterial species tested gave a positive result. By this method, reliable and rapid identification of H. **pylori** could be accomplished.

Tags: Human; Support, Non-U.S. Gov't

12484829 PMID: 12933851

A novel pathogenicity island integrated adjacent to the thrW tRNA gene of avian pathogenic Escherichia coli encodes a vacuolating autotransporter toxin.

Parreira V R; Gyles C L

Department of Pathobiology, University of Guelph, Guelph, Ontario N1G 2W1, Canada.

Infection and immunity (United States) Sep 2003, 71 (9) p5087-96, ISSN 0019-9567 Journal Code: 0246127

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

We report the complete nucleotide sequence and genetic organization of the Vat-encoding pathogenicity island (PAI) of avian pathogenic Escherichia coli strain Ec222. The 22,139-bp PAI is situated adjacent to the 3' terminus of the thrW tRNA gene, has a G+C content of 41.2%, and includes a bacteriophage SfII integrase gene, mobile genetic elements, two open reading frames with products exhibiting sequence similarity to known proteins, and several other open reading frames of unknown function. The PAI encodes an autotransporter protein, Vat (vacuolating autotransporter toxin), which induces the formation of intracellular vacuoles resulting in cytotoxic effects similar to those caused by the VacA toxin from **Helicobacter pylori** . The predicted 148.3-kDa protein product possesses the three domains that are typical of serine protease autotransporters of Enterobacteriaceae: an N-terminal signal sequence of 55 amino acids, a 111.8-kDa passenger domain containing a modified serine protease site (ATSGSG), and a C-terminal outer membrane translocator of **30** .5 **kDa** . Vat has 75% protein homology with the hemagglutinin Tsh, an autotransporter of avian pathogenic E. coli. A vat deletion mutant of Ec222 showed no virulence in respiratory and cellulitis infection models of disease in broiler chickens. We conclude that the newly described PAI and Vat may be involved in the pathogenicity of avian septicemic E. coli strain Ec222 and other avian pathogenic E. coli strains.

Tags: Support, Non-U.S. Gov't

Descriptors: *Bacterial Toxins--genetics--GE; *Escherichia coli--genetics --GE; *Escherichia coli--pathogenicity--PY; *Escherichia coli Proteins --genetics--GE; *Genes, Bacterial; Amino Acid Sequence; Animals; Bacterial Toxins--toxicity--TO; Base Sequence; Cells, Cultured; Chick Embryo; Chickens; Chromosome Mapping; DNA, Bacterial--genetics--GE; Escherichia coli Infections--etiology--ET; Escherichia coli Infections--pathology--PA; Escherichia coli Proteins--toxicity--TO; Molecular Sequence Data; Sequence Homology, Amino Acid; Vacuoles--pathology--PA; Virulence--genetics--GE

Molecular Sequence Databank No.: GENBANK/AY151282

CAS Registry No.: 0 (Bacterial Toxins); 0 (DNA, Bacterial); 0 (Escherichia coli Proteins)

Record Date Created: 20030822

Record Date Completed: 20030929

3/9/23

12300247 PMID: 12656696

Clinical and pathological implications of IgG antibody responses to Helicobacter pylori **and its virulence factors in non-ulcer dyspepsia.**

Xia H H-X; Talley N J; Blum A L; O'Morain C A; Stolte M; Bolling-Sternevald E; Mitchell H M

Department of Medicine, University of Sydney, Nepean Hospital, Penrith, NSW, Australia.

Alimentary pharmacology & therapeutics (England) Apr 1 2003, 17 (7) p935-43, ISSN 0269-2813 Journal Code: 8707234

Document type: Clinical Trial; Journal Article; Multicenter Study; Randomized Controlled Trial

Languages: ENGLISH

Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
AIM: To determine whether pre-treatment antibody response to **Helicobacter pylori** virulence factors predicts eradication success and symptom relief 12 months after triple therapy in non-ulcer dyspepsia. METHODS: H. **pylori** -positive patients with non-ulcer dyspepsia received 1-week omeprazole-based triple therapy, or omeprazole plus placebos. Symptoms were assessed using a validated Likert scale. Gastric biopsies taken before and 12 months after treatment were used for histological examination. Pre-treatment blood samples were used for the detection of anti-H. **pylori** immunoglobulin G (IgG) antibodies, and specific IgG antibodies to 19.5-, 26.5-, **30** -, 35-, 89- (VacA) and 116- **kDa** (CagA) antigens of H. **pylori** . RESULTS: IgG antibodies to the six antigens were detected in 62%, 96%, 88%, 47%, 54% and 78% of patients, respectively. The presence of antibody to 19.5-, 26.5- or **30** - **kDa** antigen was associated with an increased anti-H. **pylori** IgG absorbance index. IgG absorbance indices were greater in those with H. **pylori** eradication (vs. persistent infection). The prevalence of antibodies to the six antigens was not significantly different between those with symptom relief vs. those without. The 19.5-kDa antigen (P = 0.018) and VacA (P = 0.001) were independent risk factors for body gastritis. CONCLUSIONS: An increased pre-treatment anti-H. **pylori** IgG absorbance index may be a useful predictor of the success of eradication therapy. Although the 19.5-kDa antigen and VacA were associated with body gastritis, none of the six antigens tested predicted symptom relief after triple therapy.
Tags: Female; Human; Male
Descriptors: Dyspepsia--immunology--IM; * **Helicobacter** Infections --immunology--IM; * **Helicobacter pylori** --immunology--IM; *Immunoglobulin G--immunology--IM; Adult; Aged; Aged, 80 and over; Amoxicillin--therapeutic use--TU; Anti-Ulcer Agents--therapeutic use--TU; Antibodies, Bacterial --immunology--IM; Double-Blind Method; Dyspepsia--microbiology--MI; **Helicobacter** Infections--complications--CO; **Helicobacter** Infections --drug therapy--DT; Middle Aged; Omeprazole--therapeutic use--TU; Penicillins--therapeutic use--TU
CAS Registry No.: 0 (Anti-Ulcer Agents); 0 (Antibodies, Bacterial); 0 (Immunoglobulin G); 0 (Penicillins); 26787-78-0 (Amoxicillin); 73590-58-6 (Omeprazole)
Record Date Created: 20030326
Record Date Completed: 20030623

3/9/24
DIALOG(R)File 155:MEDLINE(R)

12256082 PMID: 12603615
Clinical application of 20 MHz endosonography and anti- Helicobacter pylori **immunoblots to predict regression of low-grade gastric MALToma by H.** pylori **eradication.**
Sheu Bor-Shyang; Shiesh Shu-Chu; Wang Jin-Town; Yang Hsiao-Bai; Lin Shih-Ting; Wu Jiunn-Jong
Department of Internal Medicine, National Cheng Kung University Medical College, Tainan, Taiwan.
Helicobacter (United States) Feb 2003, 8 (1) p36-45, ISSN 1083-4389 Journal Code: 9605411
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
AIM: We tested whether serial 20 MHz endosonography (EUS) and anti- **Helicobacter pylori** immunoblots can predict the complete regression of gastric MALToma by H. **pylori** eradication. METHODS: The serums of 17 MALToma patients, including 15 with low grade and two with high grade, were collected before therapy. Fifteen patients with low-grade MALToma and 18 nonMALToma patients, all infected with H. **pylori** , have been followed with

serum sampling, endoscopy, and EUS on enrollment, on the 2nd, 6th, and 12th months after anti-H. **pylori** therapy. All sera were tested for anti-H. **pylori** immunoblots, including 19.5, 26.5, **30** , 35, 89, 116 **KDa** (CagA), FldA. The DNAs were extracted serially from the biopsy of MALToma patients before and after therapy to perform polymerase chain reaction (PCR) for the immunoglobulin heavy-chain gene monoclonality. RESULTS: MALToma patients had higher prevalence rates of anti-FldA protein, 19.5 and **30** **KDa** antibodies of H. **pylori** ($p < 0.01$). After H. **pylori** eradication, MALToma patients had negative seroconversion of 19.5, 26.5, **30** , and 35 **KDa** antibodies ($p < 0.05$), but not in CagA and FldA. The PCR monoclonality occurred in 80% (12/15) of the MALToma patients before therapy, but did not correlate with any seroconversion of anti-H. **pylori** immunoblots after therapy ($p > 0.05$). Complete regression of MALToma was observed in 73.3% (11/15) of patients. Evaluation with 20 MHz EUS, for the initial tumor depth and its normalization on the 6th month had 90.9% sensitivity and 100% specificity to predict the complete regression. DISCUSSION: The negative seroconversions of the smaller-molecular-weight proteins, but not CagA and FldA, correlate with regression of MALToma by H. **pylori** eradication. 20 MHz EUS can effectively predict the therapeutic response of MALToma.

Tags: Female; Human; Male; Support, Non-U.S. Gov't

Descriptors: **Helicobacter** Infections--complications--CO; * **Helicobacter** Infections--microbiology--MI; * **Helicobacter** **pylori** ; *Lymphoma, Mucosa-Associated Lymphoid Tissue--complications--CO; *Lymphoma, Mucosa-Associated Lymphoid Tissue--microbiology--MI; *Stomach Neoplasms--complications--CO; *Stomach Neoplasms--microbiology--MI; Adult; Antibiotics, Combined--therapeutic use--TU; Antibodies, Bacterial--blood --BL; Case-Control Studies; Endosonography; Genes, Immunoglobulin; **Helicobacter** Infections--drug therapy--DT; **Helicobacter** Infections --ultrasonography--US; **Helicobacter** **pylori** --immunology--IM; Immunoblotting; Lymphoma, Mucosa-Associated Lymphoid Tissue --ultrasonography--US; Middle Aged; Stomach Neoplasms--ultrasonography--US

CAS Registry No.: 0 (Antibiotics, Combined); 0 (Antibodies, Bacterial)

Record Date Created: 20030226

Record Date Completed: 20030527

3/9/25

DIALOG(R)File 155:MEDLINE(R)

12008337 PMID: 12226680

Helicobacter pylori **anti-CagA antibodies: prevalence in symptomatic and asymptomatic subjects in Turkey.**

Abasiyanik M Fatih; Sander Ersan; Salih Barik A

Fatih University, Istanbul, Turkey.

Canadian journal of gastroenterology = Journal canadien de gastroenterologie (Canada) Aug 2002, 16 (8) p527-32, ISSN 0835-7900 Journal Code: 8807867

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

BACKGROUND: Several reports have shown the prevalence of anti-CagA antibodies to be associated with the development of peptic ulcer diseases, while others have indicated that there is no such association. AIM: To examine the prevalence of antibodies to CagA and other **Helicobacter** **pylori** antigens in symptomatic and asymptomatic subjects in Turkey. subjects and Methods: Sixty-six symptomatic subjects, 16 to 74 years of age, were examined for H **pylori** by biopsy-based tests and ELISA. One hundred nineteen asymptomatic subjects, 20 to 65 years of age, were also tested serologically for the presence of H **pylori** . Samples from both groups that were found to be positive for H **pylori** by ELISA were then tested by immunoblotting. RESULTS: Fifty-four (82%) symptomatic subjects and 76 (64%) asymptomatic subjects were found to be H **pylori** -positive by ELISA. Samples from 30 symptomatic subjects who were found to be H **pylori**

-positive by ELISA were analyzed by immunoblotting. Antibodies to CagA (116 kDa) antigen were detected in immunoblots of 11 of 14 (79%) with chronic gastritis, 12 of 13 (92%) with duodenal ulcer and three of three (100%) with gastric cancer. Antigens of the following molecular weights were also detected in these **30** subjects: 89 **kDa** (VacA) in 21 (70%), 37 kDa in 21 (70%), 35 **kDa** in 19 (63%), **30** **kDa** in 27 (90%) and 19.5 kDa in 19 (63%). Immunoblots of 40 ELISA-positive asymptomatic subjects showed that 33 (83%) had antibodies to CagA antigen, 26 (65%) to VacA antigen, **30** (75%) to a 37 **kDa** antigen, **30** (75%) to a 35 **kDa** antigen, 39 (98%) to a **30** **kDa** antigen and 36 (90%) to a 19.5 kDa antigen. CONCLUSIONS: Antibodies to CagA antigen were prevalent in both groups, regardless of the presence of gastroduodenal disease.

Tags: Human

Descriptors: Antibodies, Bacterial--blood--BL; * **Helicobacter** Infections --epidemiology--EP; * **Helicobacter** **pylori** --immunology--IM; Antigens, Bacterial--immunology--IM; Bacterial Proteins--immunology--IM; Enzyme-Linked Immunosorbent Assay; **Helicobacter** Infections--blood--BL; **Helicobacter** Infections--immunology--IM; Middle Aged; Sensitivity and Specificity; Seroepidemiologic Studies; Turkey--epidemiology--EP

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (cagA protein, Helicobacter pylori)

Record Date Created: 20020912

Record Date Completed: 20021119

3/9/26

DIALOG(R)File 155:MEDLINE(R)

11702098 PMID: 11876708

Specific identification of three low molecular weight membrane-associated antigens of Helicobacter pylori .

Voland P; Weeks D L; Vaira D; Prinz C; Sachs G

Department of Medicine II, Technical University of Munich, Munich, Germany.

Alimentary pharmacology & therapeutics (England) Mar 2002, 16 (3) p533-44, ISSN 0269-2813 Journal Code: 8707234

Contract/Grant No.: DK41301; DK; NIDDK; DK46917; DK; NIDDK; DK53462; DK; NIDDK

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

BACKGROUND: A large number of **Helicobacter** **pylori** proteins are antigenic, but antibodies to these proteins persist in spite of the eradication of the infection. METHODS AND RESULTS: The analysis of sera from H. **pylori** -infected and non-infected patients, before and 3 and 5 months after eradication, showed that the antibody response against unknown H. **pylori** antigens at 32, **30** , 22 and 14 **kDa** in sodium dodecylsulphate polyacrylamide gel electrophoresis decreased by > or = 60% at 3 months and > or = 70% at 5 months after treatment. Two-dimensional gel electrophoresis and mass spectrometry allowed the identification of eight proteins at these positions: neuraminyl-lactose-binding haemagglutinin precursor, 3-oxoadipate CoA-transferase subunit A, elongation factor P, peptidoglycan-associated lipoprotein precursor, hypothetical protein HP0596, adhesin-thiol peroxidase, 50S ribosomal protein L7/L12 and subunit b' of the F(0) ATP synthase. Three of these eight, expressed as recombinant proteins (32 **kDa** neuraminyl-lactose-binding haemagglutinin precursor, **30** **kDa** peptidoglycan-associated lipoprotein precursor and 22 kDa hypothetical protein HP0596), reacted specifically with sera from infected patients, while the 14 kDa 50S ribosomal protein L7/L12 cross-reacted with one out of five sera from H. **pylori** -negative patients. The other recombinant proteins did not show significant immunoreactivity. CONCLUSIONS: Four low molecular weight antigens were identified by these methods, three of which were specific. Immunoreaction with these three proteins (neuraminyl-lactose-binding haemagglutinin precursor,

peptidoglycan-associated lipoprotein precursor and hypothetical protein HP0596) could provide a serological assessment not only of H. **pylori** infection, but also of eradication.

Tags: Female; Human; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: Antibody Specificity; *Antigens, Bacterial--immunology--IM; * **Helicobacter** Infections--immunology--IM; * **Helicobacter pylori** --immunology--IM; *Membrane Proteins--immunology--IM; Aged; Antigens, Bacterial--analysis--AN; Blotting, Western; Gene Expression; **Helicobacter** Infections--drug therapy--DT; **Helicobacter pylori** --drug effects--DE; Immune Sera--immunology--IM; Isoelectric Focusing; Membrane Proteins --analysis--AN; Middle Aged; Molecular Weight; Serologic Tests; Species Specificity; Spectrum Analysis, Mass

CAS Registry No.: 0 (Antigens, Bacterial); 0 (Immune Sera); 0 (Membrane Proteins)

Record Date Created: 20020305

Record Date Completed: 20020604

3/9/27

DIALOG(R)File 155:MEDLINE(R)

11560135 PMID: 11730836

Identification of novel immunogenic proteins of Helicobacter pylori **by proteome technology.**

Utt M; Nilsson I; Ljungh A; Wadstrom T

Department of Medical Microbiology, Dermatology and Infection, University of Lund, Solvegatan 23, S-223 62 Lund, Sweden. Meeme.Utt@mmb.lu.se

Journal of immunological methods (Netherlands) Jan 1 2002, 259 (1-2) p1-10, ISSN 0022-1759 Journal Code: 1305440

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Cell surface proteins of the human gastric pathogen **Helicobacter pylori** , reference strain CCUG 17874, were extracted with acid glycine and fractionated by heparin affinity chromatography. The extracts were subsequently analysed using high-resolution two-dimensional gel electrophoresis (2-DE) and immunoblotting. Four proteins of low molecular masses (25- **30** **kDa**) stained by Coomassie R-350, were identified by peptide ESI-MS/MS sequencing after in-gel tryptic digestion. The identified proteins were recognised by sera from H. **pylori** -infected patients. Two of them are now described for the first time as immunogenic proteins of which one protein was determined to be distinct from all H. **pylori** proteins previously described. In addition, the specificity of the identified peptides was evaluated using both 1-D and 2-D immunoblotting against a panel of sera from patients with various bacterial infections. The present identification of highly specific antigens of H. **pylori** will encourage the improvement of serological diagnostic tests to diagnose and monitor H. **pylori** infection.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: Antigens, Bacterial--immunology--IM; *Bacterial Outer Membrane Proteins--immunology--IM; * **Helicobacter** Infections--immunology --IM; * **Helicobacter pylori** --immunology--IM; Antigens, Bacterial --isolation and purification--IP; Bacterial Outer Membrane Proteins --isolation and purification--IP; Cross Reactions; Electrophoresis, Gel, Two-Dimensional; **Helicobacter** Infections--blood--BL; Immunoblotting; Proteome; Serologic Tests

CAS Registry No.: 0 (Antigens, Bacterial); 0 (Bacterial Outer Membrane Proteins); 0 (Proteome)

Record Date Created: 20011203

Record Date Completed: 20020201

3/9/28

DIALOG(R)File 155:MEDLINE(R)

11517236 PMID: 11683922

Helicobacter pylori **in gastric cancer and peptic ulcer disease in a Colombian population. Strain heterogeneity and antibody profiles.**

Pineros D M; Riveros S C; Marin J D; Ricardo O; Diaz O O

Laboratory of Immunology, Instituto Nacional de Cancerologia, Santafe de Bogota, Colombia, South America.

Helicobacter (United States) Sep 2001, 6 (3) p199-206, ISSN 1083-4389 Journal Code: 9605411

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

BACKGROUND: **Helicobacter pylori** infection is associated with peptic ulcer disease and gastric cancer. The aim of this work was to describe, to compare H. **pylori** antigenic profiles, and to characterize the antibody response against Colombian strains in gastric cancer and peptic ulcer patients. MATERIALS AND METHODS: Liquid culture supernatants were used to determine the antigenic profiles of 35 H. **pylori** strains by immunoblotting using a pool of positive sera. Characterization of strains included the evaluation of cytotoxic and vacuolating activities. The serologic antibody profiles of 124 patients (54 duodenal ulcer and 70 gastric cancer) were analyzed against two native strains (Hpu24, Hpc29) and NCTC11638. Antibodies to specific antigenic bands in each strain were related with presentation. RESULTS: Differences among antigenic profiles were observed between native isolates with each serum recognizing a wide range of antigens (**30** -120 **Kd**). A 68 **Kd** band in Hpu24 strain was recognized by 50% of sera from peptic ulcer patients but not by gastric cancer sera (p =.000). The immune profiles differed according to the strain used (i.e. a given sera did not recognize the same bands in different strains). Detection of H. **pylori** in gastric mucosa was associated with the presence of antibodies against low molecular weight antigenic bands. CONCLUSIONS: The heterogeneity in the antibody response to H. **pylori** and the prevalence specific anti-H. **pylori** antibodies in a specific disease depend on the strain used as antigen. The results support the hypothesis that there may be a differential antibody response to carcinogenic and ulcerogenic strains and suggest that there are antigenic bands that could be useful as markers of disease.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: **Helicobacter** Infections--complications--CO; *Peptic Ulcer --microbiology--MI; *Stomach Neoplasms--microbiology--MI; Adult; Antibodies, Bacterial--immunology--IM; Antigens, Bacterial--immunology--IM; Colombia; Hela Cells; **Helicobacter** Infections--drug therapy--DT; **Helicobacter** Infections--immunology--IM; **Helicobacter pylori** --immunology--IM; **Helicobacter pylori** --pathogenicity--PY; Middle Aged; Peptic Ulcer--drug therapy--DT; Peptic Ulcer--immunology--IM; Peptic Ulcer--pathology--PA; Species Specificity; Stomach Neoplasms--drug therapy --DT; Stomach Neoplasms--immunology--IM; Stomach Neoplasms--pathology--PA ; Vacuoles

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial)

Record Date Created: 20011030

Record Date Completed: 20011213

3/9/29

DIALOG(R)File 155:MEDLINE(R)

11514402 PMID: 11681214

Identification of a tyrosine-phosphorylated 35 kDa carboxy-terminal fragment (p35CagA) of the Helicobacter pylori **CagA protein in phagocytic cells: processing or breakage?**

Moese S; Selbach M; Zimny-Arndt U; Jungblut P R; Meyer T F; Backert S

Max-Planck-Institut fur Infektionsbiologie, Abt., Molekulare Biologie,

Schumannstr. 20/21, D-10117 Berlin, Germany.
Proteomics (Germany) Apr 2001, 1 (4) p618-29, ISSN 1615-9853
Journal Code: 101092707
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Helicobacter pylori is a very common bacterial pathogen that causes gastric disease by inducing the infiltration of immune cells as an initial event. Virulent H. **pylori** strains express a type IV secretion system composed of several virulence (Vir) proteins encoded by the cag pathogenicity island (cag PAI). During infection of phagocytic cells (U937, Josk-M and J774A.1) we have detected a de novo tyrosine-phosphorylated protein (p35p-Tyr) with sizes of **30 kDa**, 38 **kDa** or 40 **kDa**, depending on the H. **pylori** strain. p35p-Tyr occurrence required functional virB4, virB7, virB10, virB11, virD4 and cagA (cytotoxin-associated gene A) genes encoded by the cag PAI suggesting that p35p-Tyr is a bacterial protein of variable size. We have biochemically purified p35p-Tyr from infected U937 cells. Tryptic peptides of p35p-Tyr determined by matrix-assisted laser desorption/ionization-mass spectrometry (MALDI-MS) identified the carboxy (C)-terminal part of the H. **pylori** CagA protein. Subsequent analysis by two-dimensional electrophoresis (2-DE) and immunoblotting using anti-CagA antibodies revealed the presence of three stable CagA protein species in phagocytes: (i) 130-140 kDa full-length CagA (p135CagA), (ii) a 100-105 **kDa** fragment (p100CagA) and (iii) a **30** -40 **kDa** fragment (p35CagA). Unlike p135CagA, p35CagA and p100CagA were also detected in much lower amounts in H. **pylori** without host cell contact. Therefore, breakage or processing leads to the production of p35CagA and p100CagA, a process that is enhanced after translocation into host cells. MALDI-MS data and the isoelectric point determined by both 2-DE and sequence analysis suggested that p35CagA represents the C-terminal part of CagA and p100CagA corresponds to the remaining amino (N)-terminal fragment. The possible function of CagA in host signal transduction and development of gastric disease is discussed.

Tags: Human; Support, Non-U.S. Gov't
Descriptors: Antigens, Bacterial; *Bacterial Proteins--chemistry--CH; *Bacterial Proteins--metabolism--ME; * **Helicobacter pylori** --metabolism --ME; *Peptide Fragments--chemistry--CH; *Peptide Fragments--metabolism --ME; Amino Acid Sequence; Bacterial Proteins--genetics--GE; Cell Line; Electrophoresis, Gel, Two-Dimensional; Genes, Bacterial; **Helicobacter** Infections--etiology--ET; **Helicobacter pylori** --genetics--GE; **Helicobacter pylori** --pathogenicity--PY; Immunoblotting; Molecular Sequence Data; Molecular Weight; Peptide Fragments--genetics--GE; Peptide Mapping; Phagocytes--metabolism--ME; Phagocytes--microbiology--MI; Phosphorylation; Protein Processing, Post-Translational; Proteome; Spectrometry, Mass, Matrix-Assisted Laser Desorption-Ionization; Tyrosine --metabolism--ME; Virulence
CAS Registry No.: 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (Peptide Fragments); 0 (Proteome); 0 (cagA protein, Helicobacter pylori); 55520-40-6 (Tyrosine)
Record Date Created: 20011029
Record Date Completed: 20011218

3/9/30
DIALOG(R)File 155:MEDLINE(R)

11252769 PMID: 11328362
The accuracy of serologic diagnosis of Helicobacter pylori **infection in school-aged children of mixed ethnicity.**
Tindberg Y; Bengtsson C; Bergstrom M; Granstrom M
Sachs' Department of Pediatrics, Sodersjukhuset, Karolinska Institutet, Stockholm, Sweden. Ylva.Tindberg@mep.ki.se
Helicobacter (United States) Mar 2001, 6 (1) p24-30, ISSN 1083-4389
Journal Code: 9605411

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

The present study evaluated two non-invasive diagnostic methods for H. **pylori** infection in children, i.e. an in-house ELISA using sonicated Campylobacter jejuni antigen for absorption of cross-reacting antibodies and an immunoblot kit (Helico Blot 2.0, Genelabs, Singapore). 13C -Urea breath test (13C-UBT) was used as reference METHOD: Sera and questionnaires were collected from 695/858 (81%) Swedish school children with mixed ethnic backgrounds within a cross-sectional, community-based study. Of 133 children with an ELISA OD value of > or = 0.1, all were screened with immunoblot and 107 made a 13C-UBT. The negative controls were 34/37 children from three school classes with an ELISA OD value of < 0.1 and volunteering for a 13C-UBT. An adjusted cut-off level for the ELISA of OD value 0.22 resulted in a sensitivity of 97.8%, a specificity of 95.8% and a concordance index of 97.2%. The Helico Blot 2.0 had a sensitivity of 97.8%, a specificity of 93.8% and a concordance index of 96.5%. The best concordance was seen for the 26.5 **kDa** (98.6%), **30 kDa** (95.7%) and 19.5 kDa (91.5%) antigens. The corresponding concordance index for CagA was 78%, for VacA 73.8% and for the 35kDa antigen 68.8%. A significant difference in the distribution of the 19.5 and 26.5 kDa bands but not of CagA/VacA was noted by ethnic background. With an adjusted cut-off level for the enzyme-linked immunosorbent assay (ELISA), both non-invasive methods were found to have an adequate performance in a pediatric population. The differences in antibody response patterns by ethnic background represent a caveat in the interpretation of serological studies.

Tags: Comparative Study; Female; Human; Male; Support, Non-U.S. Gov't

Descriptors: **Helicobacter** Infections--diagnosis--DI; Adolescent; Antigens, Bacterial--metabolism--ME; Bacterial Proteins--metabolism--ME; Breath Tests; Child; Enzyme-Linked Immunosorbent Assay; Evaluation Studies; **Helicobacter** Infections--ethnology--EH; **Helicobacter pylori** --metabolism--ME; Immunoblotting; Sensitivity and Specificity; Serologic Tests--methods--MT; Sweden--epidemiology--EP; Urea--analysis--AN

CAS Registry No.: 0 (Antigens, Bacterial); 0 (Bacterial Proteins); 0 (cagA protein, Helicobacter pylori); 0 (vacuolating toxin, Helicobacter pylori); 57-13-6 (Urea)

Record Date Created: 20010430
Record Date Completed: 20010802

3/9/31

DIALOG(R)File 155:MEDLINE(R)

10762496 PMID: 10882654

Antibody to heat shock protein can be used for early serological monitoring of Helicobacter pylori **eradication treatment.**

Yunoki N; Yokota K; Mizuno M; Kawahara Y; Adachi M; Okada H; Hayashi S; Hirai Y; Oguma K; Tsuji T

First Department of Internal Medicine, Okayama University Medical School, Japan.

Clinical and diagnostic laboratory immunology (UNITED STATES) Jul 2000, 7 (4) p574-7, ISSN 1071-412X Journal Code: 9421292

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Infection with **Helicobacter pylori** induces humoral immune responses against various antigens of the bacterium. Heat shock proteins (hsps) are immunodominant antigens in various diseases including H. **pylori** infection. In the present study, we measured the anti-hsp antibody titers in 42 patients with H. **pylori** -infected peptic ulcers during a bacterial eradication study. The patients were treated with a proton pump inhibitor and antimicrobial agents to eradicate the organism. Their sera were

obtained at pretreatment and at 1 month and 6 months after the eradication therapy. The titers of immunoglobulin G antibodies to the H. **pylori** hsp, whole-cell lysate, and urease (**30** - **kDa** subunit) antigens in serum were measured by a capture enzyme-linked immunosorbent assay. The levels of H. **pylori** hsp60 antibodies in sera collected 1 month after treatment had declined significantly, even when changes in the titers of antibodies to whole-cell and urease antigens were not apparent. These results suggest that measurement of antibodies to H. **pylori** hsp60 in serum is useful for the early monitoring of the effectiveness of eradication therapy.

Tags: Female; Human; Male; Support, Non-U.S. Gov't

Descriptors: Heat-Shock Proteins--immunology--IM; * **Helicobacter** Infections--immunology--IM; * **Helicobacter pylori** --immunology--IM; *Immunoglobulin G--blood--BL; Adult; Aged; Anti-Bacterial Agents --therapeutic use--TU; Biological Markers; **Helicobacter** Infections--blood --BL; **Helicobacter** Infections--drug therapy--DT; **Helicobacter pylori** --isolation and purification--IP; Immunoglobulin G--immunology--IM; Middle Aged

CAS Registry No.: 0 (Anti-Bacterial Agents); 0 (Biological Markers); 0 (Heat-Shock Proteins); 0 (Immunoglobulin G)

Record Date Created: 20000912

Record Date Completed: 20000912

3/9/32

DIALOG(R)File 155:MEDLINE(R)

10684523 PMID: 10797079

Use of digoxigenin-labelled ampicillin in the identification of penicillin-binding proteins in Helicobacter pylori .

Harris A G; Hazell S L; Netting A G

School of Microbiology and Immunology, University of New South Wales, Sydney 2052, NSW.

Journal of antimicrobial chemotherapy (ENGLAND) May 2000, 45 (5) p591-8, ISSN 0305-7453 Journal Code: 7513617

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Amoxycillin is used in current therapeutic regimens to treat the infection caused by the human gastric pathogen, **Helicobacter pylori** . The penicillin-binding proteins (PBPs) are the primary targets for the beta-lactam antibiotics, such as amoxycillin, and are involved in the terminal stages of peptidoglycan synthesis. They also play active roles in the determination and maintenance of cellular morphology. It was believed that an organism with a complex morphology, such as H. **pylori** , would have more than the three PBPs previously suggested. Using digoxigenin-labelled ampicillin (DIG-ampicillin), we report the identification of eight PBPs in H. **pylori** with masses of 72, 62, 54, 50, 44, 33.5, **30** .5 and 28 **kDa** . A smaller (21 kDa) ninth band was also detected, which may represent another PBP. However, the relatively small size of this apparent PBP raises questions as to whether this is a true PBP. In an attempt to identify the PBPs to which amoxycillin preferentially binds, amoxycillin was used in competition assays with DIG-ampicillin. It appeared that amoxycillin inhibited the binding of DIG-ampicillin to only the 72 kDa PBP. The experimental data were also compared with the seven putative PBPs identified in the two published H. **pylori** genomes, most of which correlate with the experimental data. To investigate further the properties of these PBPs, the seven putative PBP genes identified in the H. **pylori** genomes were examined. The derived amino acid sequences of the putative PBPs were examined for the three characteristic motifs found in all conventional PBPs, SXXK, SXN and KTG. We were able to determine that all of the putative PBPs had at least one of these motifs, but none possessed all three motifs with the characteristics of conventional PBPs. These findings suggest that the PBPs of H. **pylori** are unique.

Tags: Support, Non-U.S. Gov't

Descriptors: Ampicillin--metabolism--ME; *Bacterial Proteins; *Carrier Proteins--genetics--GE; *Carrier Proteins--metabolism--ME; * **Helicobacter pylori** --metabolism--ME; *Hexosyltransferases; *Muramoylpentapeptide Carboxypeptidase--genetics--GE; *Muramoylpentapeptide Carboxypeptidase --metabolism--ME; *Peptidyltransferase; Ampicillin--chemistry--CH; Binding, Competitive; Carrier Proteins--isolation and purification--IP; Digoxigenin --chemistry--CH; Digoxigenin--metabolism--ME; **Helicobacter pylori** --genetics--GE; Muramoylpentapeptide Carboxypeptidase --isolation and purification--IP

CAS Registry No.: 0 (Bacterial Proteins); 0 (Carrier Proteins); 1672-46-4 (Digoxigenin); 69-53-4 (Ampicillin)

Enzyme No.: EC 2.3.2.12 (Peptidyltransferase); EC 2.4.1.- (Hexosyltransferases); EC 3.4.16.4 (penicillin-binding protein); EC 3.4.17.8 (Muramoylpentapeptide Carboxypeptidase)

Record Date Created: 20000811

Record Date Completed: 20000811

3/9/33

DIALOG(R)File 155:MEDLINE(R)

10648027 PMID: 10759250

Serologic response to lower-molecular-weight proteins of H. pylori **is related to clinical outcome of H.** pylori **infection in Taiwan.**

Shiesh S C; Sheu B S; Yang H B; Tsao H J; Lin X Z

Department of Medical Technology, National Cheng Kung University, Tainan, Taiwan.

Digestive diseases and sciences (UNITED STATES) Apr 2000, 45 (4) p781-8, ISSN 0163-2116 Journal Code: 7902782

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: AIM; INDEX MEDICUS

The study aimed to examine the serum serological response among H. **pylori** -infected patients with various upper gastrointestinal diagnoses; to ascertain whether it could be predictive to the diagnostic outcome of dyspepsia. One hundred seventy H. **pylori** -infected patients with dyspeptic symptoms but without previous treatment were enrolled, including those with duodenal ulcer disease (N = 47), gastric ulcer (N = 23), nonulcer dyspepsia (N = 60), gastric cancer (N = 34), and MALToma (N = 6). Sera from dyspeptic patients without H. **pylori** infection (N = 33) were used as controls. During endoscopy, gastric biopsies were taken for CLO-test, histology, and culture for the detection of H. **pylori** infection, defined by a positive culture or positive results of both CLO-test and histology. Total H. **pylori** IgG antibody was tested by an ELISA method. Antibody responses to specific H. **pylori** proteins were tested by a western blotting system. Of patients with H. **pylori** -infected gastroduodenal diseases, 76.5%, 42.9%, 23.6%, 46.7%, 84.1%, 76.5%, 82.9%, and 32.4% on average, showed responses to the 116-kDa (CagA), 89-kDa (VacA), 60- **kDa** , 45- **kDa** , 35- **kDa** , **30** - **kDa** , 26.5- **kDa** , and 19.5-kDa H. **pylori** -specific proteins, respectively. A significant association was found between the serological response to 19.5-kDa and 26.5-kDa proteins and malignant outcome of H. **pylori** infection ($P<0.02$). Among patients without malignancy, the absence of a band at 19.5 kDa was statistically associated with the presence of an ulcer ($P<0.05$). The presence of serum antibody against CagA is not different between patients with ulcer and with malignancy in clinical diagnosis. The serum test for detecting antibodies against lower-molecular-weight proteins of H. **pylori** , such as those of 19.5 and 26.5 kDa, could be useful to identify H. **pylori** -infected patients at risk of peptic ulcer or malignancy.

Tags: Female; Human; Male; Support, Non-U.S. Gov't

Descriptors: Antibodies, Bacterial--blood--BL; *Bacterial Proteins --metabolism--ME; *Dyspepsia--etiology--ET; * **Helicobacter** Infections --immunology--IM; * **Helicobacter pylori** --immunology--IM; Bacterial Proteins--chemistry--CH; Blotting, Western; Case-Control Studies; Dyspepsia

--microbiology--MI; Enzyme-Linked Immunosorbent Assay; **Helicobacter** Infections--complications--CO; **Helicobacter** Infections--microbiology --MI; Immunoglobulin G--blood--BL; Lymphoma, Mucosa-Associated Lymphoid Tissue--complications--CO; Middle Aged; Molecular Weight; Peptic Ulcer --complications--CO; Peptic Ulcer--microbiology--MI; Stomach Neoplasms --complications--CO; Taiwan
CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Bacterial Proteins); 0 (Immunoglobulin G)
Record Date Created: 20000420
Record Date Completed: 20000420

3/9/34
DIALOG(R)File 155:MEDLINE(R)

10593906 PMID: 10699497
Helicobacter pylori **-antigen-binding fragments expressed on the filamentous M13 phage prevent bacterial growth.**
Cao J; Sun Y; Berglindh T; Mellgard B; Li Z; Mardh B; Mardh S
Department of Biomedicine and Surgery, Division of Cell Biology, Faculty of Health Sciences, Linkoping University, Linkoping, Sweden.
Biochimica et biophysica acta (NETHERLANDS) Mar 6 2000, 1474 (1) p107-13, ISSN 0006-3002 Journal Code: 0217513
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Colonization of the human stomach by **Helicobacter pylori** is associated with the development of gastritis, duodenal ulcer, mucosa-associated lymphoid tissue (MALT) lymphoma, and gastric cancer. H. **pylori** -antigen-binding single-chain variable fragments (ScFv) were derived from murine hybridomas producing monoclonal antibodies and expressed as a g3p-fusion protein on a filamentous M13 phage. The recombinant ScFv-phage reacted specifically with a **30 - kDa** monomeric protein of a H. **pylori** surface antigen preparation and by means of immunofluorescence microscopy the phage was shown to bind to both the spiral and coccoid forms of the bacterium. In vitro, the recombinant phage exhibited a bacteriocidal effect and inhibited specifically the growth of all the six strains of H. **pylori** tested. When H. **pylori** was pretreated with the phage 10 min before oral inoculation of mice, the colonization of the mouse stomachs by the bacterium was significantly reduced ($P<0.01$). The results suggest that genetic engineering may be used to generate therapy-effective phages.
Tags: Support, Non-U.S. Gov't
Descriptors: Bacteriophage M13--immunology--IM; * **Helicobacter pylori** --immunology--IM; Animals; Antibodies, Monoclonal--immunology--IM; Bacteriophage M13--genetics--GE; Disease Models, Animal; Enzyme-Linked Immunosorbent Assay; Genetic Engineering; **Helicobacter** Infections --prevention and control--PC; **Helicobacter pylori** --genetics--GE; **Helicobacter pylori** --growth and development--GD; Immunoglobulin Variable Region--genetics--GE; Immunoglobulin Variable Region--immunology --IM; Mice; Microscopy, Fluorescence
CAS Registry No.: 0 (Antibodies, Monoclonal); 0 (Immunoglobulin Variable Region)
Record Date Created: 20000504
Record Date Completed: 20000504

3/9/35
DIALOG(R)File 155:MEDLINE(R)

10585723 PMID: 10693997
Immunoblotting and serology for diagnosis of Helicobacter pylori **infection in children.**

Raymond J; Sauvestre C; Kalach N; Bergeret M; Dupont C
Microbiology Service, Hopital St. Vincent de Paul, Paris, France. j.raymond@svp.ap-hop-paris.fr
Pediatric infectious disease journal (UNITED STATES) Feb 2000, 19 (2) p118-21, ISSN 0891-3668 Journal Code: 8701858
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
BACKGROUND: The easiest way to identify the presence of current or past **Helicobacter pylori** infection is to test for antibodies. The aim of this study was to compare an enzyme-linked immunosorbent assay (ELISA) technique based on the detection of IgG antibodies directed against a global antigenic preparation with immunoblotting based on the analysis of IgG antibody reactivity to separate proteins. METHODS: Sera were collected from 80 children (mean age, 9.9 +/- 4.3 years). The reference tests were microbiologic and histologic examination of gastric biopsies obtained at upper endoscopy. RESULTS: The immunoblotting was more sensitive (100%) and specific (88%) than ELISA (96 and 79%, respectively) in the evaluation of H. **pylori** infection in children. Its positive predictive value was 92%, and its negative predictive value was 100%. The best performance index of immunoreactive bands to detect antibodies was obtained with the 26- **kDa** (88.7%), **30** - **kDa** (77.5%) and 19.5-kDa (70%) antigens. Antibodies by immunoblot technique against the CagA antigen were present in 43.1% of children. CONCLUSION: Immunoblotting is highly sensitive and more specific than ELISA in children and provides additional information about the full serologic profile. Immunoblotting may therefore be a useful complement to serology, particularly in cases with doubtful ELISA results.
Tags: Comparative Study; Human
Descriptors: Antibodies, Bacterial--blood--BL; *Enzyme-Linked Immunosorbent Assay; * **Helicobacter** Infections--diagnosis--DI; * **Helicobacter pylori** --immunology--IM; *Immunoblotting; *Immunoglobulin G --blood--BL; Adolescent; Child; **Helicobacter** Infections--microbiology--MI ; Predictive Value of Tests; Reagent Kits, Diagnostic; Sensitivity and Specificity; Serologic Tests
CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Immunoglobulin G); 0 (Reagent Kits, Diagnostic)
Record Date Created: 20000323
Record Date Completed: 20000323

3/9/36
DIALOG(R)File 155:MEDLINE(R)

10012817 PMID: 8132346
Immunobiological activities of Helicobacter pylori **porins.**
Tufano M A; Rossano F; Catalanotti P; Liguori G; Capasso C; Ceccarelli M T; Marinelli P
Istituto di Microbiologia, Seconda Universita di Napoli, Italy.
Infection and immunity (UNITED STATES) Apr 1994, 62 (4) p1392-9, ISSN 0019-9567 Journal Code: 0246127
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Studies were carried out on some biological activities of **Helicobacter pylori** porins in vitro. We extracted and purified a porin with an apparent molecular mass of **30** **kDa** . Human polymorphonuclear leukocytes preincubated with H. **pylori** porins showed a decrease of chemotaxis, of adherence to nylon wool, and of chemiluminescence. Used as chemotaxins in place of zymosan-activated serum or as chemotaxinogens in place of zymosan, the porins induced polymorphonuclear leukocyte migration. Human monocytes and lymphocytes cultivated in the presence of H. **pylori** porins released cytokines. Release of the various cytokines studied was obtained with

differentiated kinetics and at various porin concentrations. Starting only 3 h after culture, tumor necrosis factor alpha is released quickly, reaching a peak at 18 h, at a porin concentration of 1 microgram/ml/10(6) cells. Interleukin-6 (IL-6) appears later, with a peak at 10 micrograms/ml/10(6) cells, while IL-8 is released after 6 h of culture, with a peak at 24 h, at a porin concentration of 10 micrograms/ml/10(6) cells, while IL-8 is released after 6 h of culture, with a peak at 24 h, at a porin concentration of 10 micrograms/ml/10(6) cells. Lymphocytes stimulated by H. **pylori** porins release gamma interferon after 18 h of culture at higher concentrations of porins (20 micrograms/ml/10(6) cells). Granulocyte macrophage colony-stimulating factor is released from 6 to 48 h at a concentration of 1 microgram/ml/10(6) cells, while both IL-3 and IL-4 are released after 18 h of culture at different porin concentrations (0.1 and 1 microgram/ml/10(6) cells, respectively). Our results lead us to think that during H. **pylori** infection, surface components, porins in particular, are able to induce a series of chain reactions ranging from the inflammatory to the immunological responses.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: **Helicobacter pylori** --pathogenicity--PY; *Porins --pharmacology--PD; Chemotaxis, Leukocyte--drug effects--DE; Granulocyte-Macrophage Colony-Stimulating Factor--secretion--SE; Interleukins--secretion --SE; Lymphocytes--drug effects--DE; Lymphocytes--secretion--SE; Monocytes --drug effects--DE; Monocytes--secretion--SE; Neutrophils--drug effects --DE; Neutrophils--immunology--IM; Tumor Necrosis Factor--secretion--SE

CAS Registry No.: 0 (Interleukins); 0 (Porins); 0 (Tumor Necrosis Factor); 83869-56-1 (Granulocyte-Macrophage Colony-Stimulating Factor)

Record Date Created: 19940421

Record Date Completed: 19940421

3/9/37

DIALOG(R)File 155:MEDLINE(R)

09989985 PMID: 8112521

Inhibition of Helicobacter pylori **glycosulfatase activity towards human gastric sulfomucin by a gastroprotective agent, sulglycotide.**

Murty V L; Piotrowski J; Czajkowski A; Slomiany A; Slomiany B L

Research Center, University of Medicine and Dentistry of New Jersey, Newark 07103-2400.

General pharmacology (ENGLAND) Nov 1993, 24 (6) p1463-6, ISSN 0306-3623 Journal Code: 7602417

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

1. A glycosulfatase activity towards human gastric sulfomucin was identified in the extracellular material elaborated by **Helicobacter pylori** , a pathogen implicated in the etiology of gastric disease. 2. The purified enzyme displayed an apparent molecular weight of **30 kDa** , and exhibited maximum activity at pH 5.7 in the presence of 0.3% Triton X-100 and 100 mM CaCl2. 3. The H. **pylori** glycosulfatase activity towards human gastric sulfomucin was inhibited by a gastroprotective agent, sulglycotide. The inhibitory effect was proportional to the concentration of sulglycotide up to 20 micrograms/ml, at which a 98% decrease in mucin desulfation occurred. However, the drug lost the inhibitory effect following its chemical desulfation. 4. The results demonstrate that sulglycotide is a potent inhibitor of H. **pylori** glycosulfatase and, hence, may be of value in the treatment of gastric disease associated with this bacterial infection.

Tags: Human

Descriptors: Anti-Ulcer Agents--pharmacology--PD; * **Helicobacter pylori** --enzymology--EN; *Mucins--metabolism--ME; *Sialoglycoproteins--pharmacology--PD; *Sulfatases--antagonists and inhibitors--AI; Gastric Mucosa --metabolism--ME; Proteins--metabolism--ME; Sulfatases--metabolism--ME; Sulfur Radioisotopes--diagnostic use--DU

CAS Registry No.: 0 (Anti-Ulcer Agents); 0 (Mucins); 0 (Proteins); 0 (Sialoglycoproteins); 0 (Sulfur Radioisotopes); 0 (sulfomucin); 54182-59-1 (sulglicotide)
Enzyme No.: EC 3.1.6. (Sulfatases); EC 3.1.6.3 (glycosulphatase)
Record Date Created: 19940331
Record Date Completed: 19940331

3/9/38
DIALOG(R)File 155:MEDLINE(R)

09875634 PMID: 8225576
Correlation between vacuolating cytotoxin production by Helicobacter pylori **isolates in vitro and in vivo.**
Cover T L; Cao P; Lind C D; Tham K T; Blaser M J
Department of Medicine, Vanderbilt University School of Medicine, Nashville, Tennessee 37232-2605.
Infection and immunity (UNITED STATES) Dec 1993, 61 (12) p5008-12, ISSN 0019-9567 Journal Code: 0246127
Contract/Grant No.: R29 DK45293-01; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Approximately 50 to 60% of **Helicobacter pylori** isolates produce a vacuolating cytotoxin in vitro. To assess cytotoxin production in vivo, we sought to determine whether infection with a Tox+ H. **pylori** strain is associated with the presence of serum antitoxin antibodies. H. **pylori** isolates and serum samples were obtained from 30 patients, and serum samples were obtained from 20 uninfected patients as controls. Sera were tested by enzyme-linked immunosorbent assay for reactivity with the purified 87- **kDa** vacuolating cytotoxin, and the **30** H. **pylori** isolates were tested for vacuolating cytotoxin production. Supernatants from 14 (47%) of the 30 H. **pylori** isolates induced vacuolation of HeLa cells. Sera from the 30 H. **pylori** -infected patients reacted with the purified 87-kDa cytotoxin to a greater extent than sera from the uninfected controls for both immunoglobulin G (IgG) and IgA classes ($P = 0.0004$ and $P < 0.0001$, respectively). Serum IgG and IgA responses to the purified 87-kDa cytotoxin were higher among the 14 patients infected with Tox+ strains than among the 16 patients infected with Tox- strains (mean optical densities +/- standard errors of the means of 0.603 +/- 0.11 versus 0.234 +/- 0.07 [$P = 0.005$] and 0.644 +/- 0.12 versus 0.341 +/- 0.08 [$P = 0.04$] for IgG and IgA, respectively). Infection with a Tox+ strain compared with a Tox- strain was associated with increased antral polymorphonuclear leukocyte inflammation scores ($P = 0.04$). These data indicate that cytotoxin production by H. **pylori** isolates in vitro correlates with cytotoxin production in vivo and that infection with Tox+ H. **pylori** isolates may be associated with increased antral mucosal polymorphonuclear leukocyte infiltration.
Tags: Human; In Vitro; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
Descriptors: Bacterial Proteins--biosynthesis--BI; *Bacterial Toxins --biosynthesis--BI; *Cytotoxins--biosynthesis--BI; * **Helicobacter pylori** --metabolism--ME; Antibodies, Bacterial--blood--BL; Bacterial Proteins --immunology--IM; Bacterial Toxins--immunology--IM; Cytotoxins--immunology --IM; **Helicobacter** Infections--immunology--IM; **Helicobacter** Infections --microbiology--MI; **Helicobacter pylori** --immunology--IM; **Helicobacter pylori** --isolation and purification--IP; Immunoglobulin A--biosynthesis --BI; Immunoglobulin G--biosynthesis--BI
CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Bacterial Proteins); 0 (Bacterial Toxins); 0 (Cytotoxins); 0 (Immunoglobulin A); 0 (Immunoglobulin G); 0 (vacuolating toxin, Helicobacter pylori)
Record Date Created: 19931222
Record Date Completed: 19931222

3/9/39
DIALOG(R)File 155:MEDLINE(R)

09698213 PMID: 8487294
Ultrastructure and biochemical studies of the flagellar sheath of Helicobacter pylori .
Geis G; Suerbaum S; Forsthoff B; Leying H; Opferkuch W
Department of Medical Microbiology and Immunology, Ruhr-Universitat Bochum, Germany.
Journal of medical microbiology (ENGLAND) May 1993, 38 (5) p371-7, ISSN 0022-2615 Journal Code: 0224131
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Helicobacter pylori flagellar sheaths were isolated by sucrose density-gradient centrifugation and analysed by electronmicroscopy, SDS-PAGE and gas-liquid chromatography. Electronmicroscopy of thin sections of flagella showed an internal electron-dense filament and a surrounding flagellar sheath with the typical bilayer structure of a membrane. The flagellar filaments could be disintegrated by acid treatment and the resulting isolated flagellar sheaths formed vesicles, sometimes with characteristic structures. Centrifugation of flagellar preparations after acid treatment resulted in the enrichment of flagellar sheaths in the pellet. SDS-PAGE analysis of the pellet showed a reduction of the flagellin band and a number of protein bands of 150, 76, 67, 65, 53, 51, 49, 29.5, 18, 17 and 16 kDa. However, there were no major protein bands characteristic for the sheath. Differences between the protein profiles of Sarkosyl-insoluble membranes and flagellar sheaths appeared in the lower M(r) range of **30** -14 **kDa** . Major fatty acids of isolated flagellar sheaths were C 14:0, C 19:0 cyc, C 18:0, and the LPS-specific fatty acids 3-OH C 16:0 and 3-OH C 18:0. The results demonstrate that the flagellar sheaths of H. **pylori** are membranes and contain LPS and proteins.

Descriptors: Flagella--chemistry--CH; * **Helicobacter pylori** --chemistry --CH; Centrifugation, Density Gradient; Chromatography, Gas; Electrophoresis, Polyacrylamide Gel; Fatty Acids--analysis--AN; Flagella --ultrastructure--UL; **Helicobacter pylori** --ultrastructure--UL; Microscopy, Electron
CAS Registry No.: 0 (Fatty Acids)
Record Date Created: 19930610
Record Date Completed: 19930610

3/9/40
DIALOG(R)File 155:MEDLINE(R)

09529272 PMID: 1452359
Purification and characterization of the urease enzymes of Helicobacter **species from humans and animals.**
Turbett G R; Hoj P B; Horne R; Mee B J
Department of Microbiology, University of Western Australia, Queen Elizabeth II Medical Centre, Nedlands.
Infection and immunity (UNITED STATES) Dec 1992, 60 (12) p5259-66, ISSN 0019-9567 Journal Code: 0246127
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

The urease enzymes of **Helicobacter pylori** , H. mustelae, H. felis, and H. nemestrinae have been purified to homogeneity by affinity chromatography and characterized. The native urease enzymes of the four organisms were found to be almost identical, with a pI of 6.1 and molecular masses of 480 to 500 kDa, as determined by electrophoretic mobility in nondenaturing

polyacrylamide gels. Transmission electron microscopy of the native urease showed it to be a molecule approximately 13 nm in diameter, with hexagonal symmetry. Denaturation studies indicated that each urease enzyme molecule was composed of two nonidentical subunits with molecular masses of approximately 64 and **30 kDa** . The subunits were present in a 1:1 ratio, suggesting a hexameric stoichiometry for the native molecule. The predicted molecular mass of H. **pylori** urease, based on subunit molecular weight and stoichiometry, is 568 kDa. N-terminal amino acid sequencing of the enzyme subunits from the four species revealed high levels of homology. The large subunits (UreB) were found to be 92 to 100% homologous, and the small subunits (UreA) were 75 to 95% homologous over the first 12 to 20 residues. The high degree of homology suggests a common ancestral origin and an important role for the urease enzymes of these organisms.

Tags: Human

Descriptors: **Helicobacter** --enzymology--EN; *Urease --isolation and purification--IP; Amino Acid Sequence; Animals; Isoelectric Point; Macaca; Macaca fascicularis; Macaca mulatta; Microscopy, Electron; Molecular Sequence Data; Molecular Weight; Papio; Swine

Enzyme No.: EC 3.5.1.5 (Urease)

Record Date Created: 19930104

Record Date Completed: 19930104

3/9/41

DIALOG(R)File 155:MEDLINE(R)

09427890 PMID: 1381553

Glycosulfatase activity of H. pylori **toward human gastric mucin: effect of sucralfate.**

Slomiany B L; Murty V L; Piotrowski J; Grabska M; Slomiany A

Research Center, New Jersey Dental School, University of Medicine and Dentistry of New Jersey, Newark.

American journal of gastroenterology (UNITED STATES) Sep 1992, 87 (9) p1132-7, ISSN 0002-9270 Journal Code: 0421030

Contract/Grant No.: AA05858-11; AA; NIAAA; DK31684-15; DK; NIDDK

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Colonization of gastric mucosa by **Helicobacter pylori** , a bacterium implicated in the etiology of gastric disease, involves the cell surface sulfated glycosphingolipid receptors for the attachment. Evidence has also been obtained recently that sulfated mucus glycoproteins have the ability to interfere with this process. Here, we show that H. **pylori** displays glycosulfatase activity, and report the specificity of this enzyme toward gastric mucosal sulfated glycoproteins and glycolipids. With 35S-labeled human gastric sulfated mucin as substrate, the enzyme activity was identified in the extracellular material elaborated by the bacterium. The glycosulfatase exhibited maximum activity at pH 5.7 in the presence of Triton X-100 and CaCl2, and gave on SDS-PAGE a protein band of **30 kDa** . Specificity studies revealed that the enzyme effectively caused desulfation of N-acetylglucosamine-6-sulfate and galactose-6-sulfate present in carbohydrate chains of gastric mucins, as well as that of glucose-6-sulfate, a constituent of mucus glyceroglucolipids. However, the H. **pylori** glycosulfatase was ineffective toward galactosyl- and lactosylceramide sulfates which serve as receptors for this bacterium attachment and contain the sulfate ester group at C-3 of galactose. The glycosulfatase activity toward human sulfated gastric mucin was inhibited by sucralfate. The inhibitory effect was proportional to the concentration of sucralfate up to 120 micrograms/ml, at which a 78% decrease in mucin desulfation occurred. The results demonstrate that H. **pylori** , through its glycosulfatase activity, affects the sulfated mucin and glyceroglucolipid content of the protective mucus layer, and that antiucler drug sucralfate is able to counteract the detrimental action of this enzyme.

Tags: Human; Support, U.S. Gov't, P.H.S.

Descriptors: Gastric Mucin--metabolism--ME; * **Helicobacter pylori** --enzymology--EN; *Sucralfate--pharmacology--PD; *Sulfatases--metabolism --ME; Substrate Specificity; Sulfatases--antagonists and inhibitors--AI
CAS Registry No.: 0 (Gastric Mucin); 54182-58-0 (Sucralfate)
Enzyme No.: EC 3.1.6. (Sulfatases); EC 3.1.6.3 (glycosulphatase)
Record Date Created: 19921008
Record Date Completed: 19921008

3/9/42
DIALOG(R)File 155:MEDLINE(R)

09400073 PMID: 1500174
Characteristics of Helicobacter pylori **variants selected for urease deficiency.**
Perez-Perez G I; Olivares A Z; Cover T L; Blaser M J
Department of Medicine, Vanderbilt University School of Medicine, Nashville, Tennessee 37232-2605.
Infection and immunity (UNITED STATES) Sep 1992, 60 (9) p3658-63, ISSN 0019-9567 Journal Code: 0246127
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
The urease of **Helicobacter pylori** is suspected to play a role in the pathogenesis of gastritis. Although all clinical isolates of H. **pylori** are urease positive (U+), we have selected and characterized several spontaneously arising urease-negative (U-) variants from wild-type strain 60190. Urease-negative variants were identified by growth in medium containing 60 mM urea and arose at a frequency of 10(-5) to 10(-6). The urease activity of the wild-type strain inhibited growth of this strain in the presence of 60 mM urea. U- variants retained the U- phenotype for more than 100 passages on medium with or without urea. The urease activities of the original U+ and derived U- cells were 9.55 to 16.7 and 0.01 to 0.17 U/mg of protein, respectively. Colonial growth and other biochemical characteristics were identical for the strains. U- variants showed three classes of whole-cell sodium dodecyl sulfate-polyacrylamide gel electrophoresis profiles: (i) identical to U+; (ii) change in the migration of the 61-kDa urease subunit; and (iii) lack of 61- and **30 - kDa** subunits. These differences were confirmed by immunoblotting and by protein separation using fast protein liquid chromatography. The U+ strain but not U- variants tolerated exposure to pH 4.0 for 60 min in the presence of urea. Supernatants of the U+ strain and U- variants contained vacuolating cytotoxin activity for HeLa cells in similar titers. By enzyme-linked immunosorbent assay, human serum samples recognized water extract from the U+ strain significantly better than extract from a U- variant lacking urease subunits. In conclusion, this study demonstrates that U- H. **pylori** variants may arise spontaneously, that urease activity enhances survival at acid pH, and that urease and cytotoxin activities are disparate phenotypes.
Tags: Human; Support, U.S. Gov't, Non-P.H.S.
Descriptors: **Helicobacter pylori** --enzymology--EN; *Urease--deficiency --DF; Bacterial Proteins--isolation and purification--IP; Cytotoxins --toxicity--TO; Hela Cells; **Helicobacter pylori** --immunology--IM; **Helicobacter pylori** --pathogenicity--PY; Hydrogen-Ion Concentration; Vacuoles
CAS Registry No.: 0 (Bacterial Proteins); 0 (Cytotoxins)
Enzyme No.: EC 3.5.1.5 (Urease)
Record Date Created: 19920917
Record Date Completed: 19920917

3/9/43
DIALOG(R)File 155:MEDLINE(R)

09399940 PMID: 1500074

Isolation of H. pylori **proteins and support for their suitability in serologic diagnoses]**

Isolierung von H. **pylori** -Proteinen und Beurteilung ihrer Eignung fur den Einsatz in serologischen Diagnosesystemen.

Bassler H M; Dreher R M; Wolf K

Institut fur Mikrobiologie und Weinforschung, Universitat Mainz.

Immunitat und Infektion (GERMANY) Jul 1992, 20 (3) p103-6, ISSN 0340-1162 Journal Code: 7505519

Document type: Journal Article ; English Abstract

Languages: GERMAN

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

H. **pylori** -proteins were separated using gel chromatographic methods. These antigens were tested for their suitability to detect H. **pylori** -specific antibodies. A complex of two proteins (62 **kDa** and **30 kDa**) was a strong and specific antigen. A third protein (13 kDa) was a good but nonspecific antigen. Concerning these facts we compared two often used antigen preparations for serodiagnosing H. **pylori** -specific antibodies (acid-glycine preparation and sarcosyl-insoluble outer membrane proteins). The sarcosyl-insoluble material contains more specific antigens and lower levels of nonspecific proteins compared to the acid-glycine preparation. Based on these results we conclude that the outer membrane preparation seems to be more suitable for the serodiagnosing of H. **pylori** -specific antibodies.

Tags: Human

Descriptors: Antibodies, Bacterial--analysis--AN; *Antigens, Bacterial --isolation and purification--IP; *Bacterial Proteins--immunology--IM; * **Helicobacter** Infections--diagnosis--DI; * **Helicobacter pylori** --immunology--IM; Bacterial Proteins--isolation and purification--IP; Enzyme-Linked Immunosorbent Assay; Molecular Weight; Serologic Tests

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Antigens, Bacterial); 0 (Bacterial Proteins)

Record Date Created: 19920916

Record Date Completed: 19920916

3/9/44

09159728 PMID: 1732414

Surface proteins from Helicobacter pylori **exhibit chemotactic activity for human leukocytes and are present in gastric mucosa.**

Mai U E; Perez-Perez G I; Allen J B; Wahl S M; Blaser M J; Smith P D

Cellular Immunology Section, National Institute of Dental Research, National Institutes of Health, Bethesda, Maryland 20892.

Journal of experimental medicine (UNITED STATES) Feb 1 1992, 175 (2) p517-25, ISSN 0022-1007 Journal Code: 2985109R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The mechanism by which **Helicobacter pylori** , a noninvasive bacterium, initiates chronic antral gastritis in humans is unknown. We now show that H. **pylori** releases products with chemotactic activity for monocytes and neutrophils. This chemotactic activity was inhibited by antisera to either H. **pylori** whole bacteria or H. **pylori** -derived urease. Moreover, surface proteins extracted from H. **pylori** and purified H. **pylori** urease (a major component of the surface proteins) exhibited dose-dependent, antibody-inhibitable chemotactic activity. In addition, a synthetic 20-amino acid peptide from the NH2-terminal portion of the 61- **kD** subunit, but not the **30 - kD** subunit, of urease exhibited chemotactic activity for monocytes and neutrophils, localizing the chemotactic activity, at least in part, to the NH2 terminus of the 61-kD subunit of urease. The ability of

leukocytes to chemotax to H. **pylori** surface proteins despite formyl-methionyl-leucyl-phenylalanine (FMLP) receptor saturation, selective inhibition of FMLP-mediated chemotaxis, or preincubation of the surface proteins with antiserum to FMLP indicated that the chemotaxis was not FMLP mediated. Finally, we identified H. **pylori** surface proteins and urease in the lamina propria of gastric antra from patients with H. **pylori** -associated gastritis but not from uninfected subjects. These findings suggest that H. **pylori** gastritis is initiated by mucosal absorption of urease, which expresses chemotactic activity for leukocytes by a mechanism not involving N-formylated oligopeptides.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: Bacterial Outer Membrane Proteins--immunology--IM; *Chemotaxis, Leukocyte--immunology--IM; *Gastric Mucosa--microbiology--MI; * **Helicobacter pylori** --immunology--IM; Antibodies, Bacterial--immunology --IM; Gastric Mucosa--immunology--IM; Gastritis--immunology--IM; Gastritis --microbiology--MI; **Helicobacter** Infections--immunology--IM; **Helicobacte r pylori** --enzymology--EN; Monocytes--immunology--IM; N-Formylmethionine Leucyl-Phenylalanine--immunology--IM; Neutrophils--immunology--IM; **Pyloric** Antrum--immunology--IM; Urease--immunology--IM

CAS Registry No.: 0 (Antibodies, Bacterial); 0 (Bacterial Outer Membrane Proteins); 59880-97-6 (N-Formylmethionine Leucyl-Phenylalanine)

Enzyme No.: EC 3.5.1.5 (Urease)

Record Date Created: 19920227

Record Date Completed: 19920227

3/9/45

DIALOG(R)File 155:MEDLINE(R)

08775820 PMID: 2276611

Identification of flagellar and associated polypeptides of Helicobacter **(formerly Campylobacter)** pylori .

Luke C J; Kubiak E; Cockayne A; Elliott T S; Penn C W

School of Biological Sciences, University of Birmingham, U.K.

FEMS microbiology letters (NETHERLANDS) Sep 1 1990, 59 (1-2) p225-30 , ISSN 0378-1097 Journal Code: 7705721

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Flagella of **Helicobacter pylori** were isolated from intact organisms by shearing and differential centrifugation. Treatment of the flagella with the detergent Triton X-100 removed the flagellar sheath, which was confirmed by electron microscopy, and the remaining naked flagella were shown by sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE) to consist primarily of a single 54 kilodalton (kDa) polypeptide. This was confirmed by immunogold labelling and electron microscopy of detergent treated whole organisms, using a mouse antiserum specific for the 54 kDa polypeptide. Polypeptides solubilised from crude flagellar preparations by detergent treatment were found to have molecular weights of 26, **30** , 58, 62, 66 and 80 **kDa** . These polypeptides are possible components of the flagellar sheath and they may represent outer membrane proteins, based on the assumption that the flagellar sheath is related in composition to the outer membrane of the organism. Analysis and definition of these components of the surface structures of the organism are important in understanding the interaction between the organism and its host in pathogenesis.

Tags: Support, Non-U.S. Gov't

Descriptors: Bacterial Proteins--analysis--AN; *Flagella--chemistry--CH; * **Helicobacter pylori** --ultrastructure--UL; *Peptides--analysis--AN; Electrophoresis, Polyacrylamide Gel; **Helicobacter pylori** --analysis--AN; Microscopy, Electron; Molecular Weight

CAS Registry No.: 0 (Bacterial Proteins); 0 (Peptides)

Record Date Created: 19910307

Record Date Completed: 19910307

3/9/46
DIALOG(R)File 155:MEDLINE(R)

08540616 PMID: 2188975
Purification and characterization of urease from Helicobacter pylori .

Dunn B E; Campbell G P; Perez-Perez G I; Blaser M J
Laboratory Service, Denver Veterans Administration Medical Center, Colorado 80220.
Journal of biological chemistry (UNITED STATES) Jun 5 1990, 265 (16) p9464-9, ISSN 0021-9258 Journal Code: 2985121R
Contract/Grant No.: BRSG-05357; RS; DRS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Urease was purified 112-fold to homogeneity from the microaerophilic human gastric bacterium, **Helicobacter pylori** . The urease isolation procedure included a water extraction step, size exclusion chromatography, and anion exchange chromatography. The purified enzyme exhibited a Km of 0.3 +/- 0.1 mM and a Vmax of 1,100 +/- 200 mumols of urea hydrolyzed/min/mg of protein at 22 degrees C in 31 mM Tris-HCl, pH 8.0. The isoelectric point was 5.99 +/- 0.03. Molecular mass estimated for the native enzyme was 380,000 +/- **30** ,000 **daltons** , whereas subunit values of 62,000 +/- 2,000 and 30,000 +/- 1,000 were determined. The partial amino-terminal sequence (17 residues) of the large subunit of H. **pylori** urease (Mr = 62,000) was 76% homologous with an internal sequence of the homohexameric jack bean urease subunit (Mr = 90,770; Takashima, K., Suga, T., and Mamiya, G. (1988) Eur. J. Biochem. 175, 151-165) and was 65% homologous with amino-terminal sequences of the large subunits of heteropolymeric ureases from Proteus mirabilis (Mr = 73,000) and from Klebsiella aerogenes (Mr = 72,000; Mobley, H. L. T., and Hausinger, R. P. (1989) Microbiol. Rev. 53, 85-108). The amino-terminal sequence (20 residues) of the small subunit of H. **pylori** urease (Mr = 30,000) was 65 and 60% homologous with the amino-terminal sequences of the subunit of jack bean urease and with the Mr = 11,000 subunit of P. mirabilis urease (Jones, B. D., and Mobley, H. L. T. (1989) J. Bacteriol. 171, 6414-6422), respectively. Thus, the urease of H. **pylori** shows similarities to ureases found in plants and other bacteria. When used as antigens in an enzyme-linked immunosorbent assay, neither purified urease nor an Mr = 54,000 protein that co-purified with urease by size exclusion chromatography was as effective as crude preparations of H. **pylori** proteins at distinguishing sera from persons known either to be infected with H. **pylori** or not.
Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
Descriptors: *Campylobacter--enzymology--EN; *Urease --isolation and purification--IP; Amino Acid Sequence; Blotting, Western; Chromatography, Gel; Chromatography, Ion Exchange; Isoelectric Point; Kinetics; Klebsiella pneumoniae--enzymology--EN; Molecular Sequence Data; Molecular Weight; Proteus mirabilis--enzymology--EN; Sequence Homology, Nucleic Acid; Urea --metabolism--ME; Urease--metabolism--ME
CAS Registry No.: 57-13-6 (Urea)
Enzyme No.: EC 3.5.1.5 (Urease)
Record Date Created: 19900702
Record Date Completed: 19900702

3/9/47
DIALOG(R)File 155:MEDLINE(R)

06944942 PMID: 3932277
Potential initiating and promoting activities of diacetyl and glyoxal in

rat stomach mucosa.

Furihata C; Yoshida S; Matsushima T

Japanese journal of cancer research - Gann (JAPAN) Sep 1985, 76 (9) p809-14, ISSN 0910-5050 Journal Code: 8509412

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The potential initiating and promoting activities in the rat glandular stomach of the dicarbonyl compounds diacetyl (DA) and glyoxal (G), which are found in various heated foods, were studied. Administration of DA at doses of 300 to 1500 mg/kg body weight and of G at doses of 150 to 400 mg/kg body weight by gastric intubation to male F344 rats induced up to 100-fold increase in ornithine decarboxylase activity (formation of 195 pmol CO2/ **30** min/mg protein by **DA** and 302 pmol CO2/ **30** min/mg protein by G) with maxima after 16 hr. These treatments also induced a more than 10-fold increase in DNA synthesis (incorporation of 11,400 dpm of [3H]dThd/microgram DNA by DA and 15,100 dpm of [3H]dThd/microgram DNA by G) with maxima after 16 hr, and induced apparent unscheduled DNA synthesis in the **pyloric** mucosa of the stomach within 3 hr after administration. These results suggest that DA and G have potential tumor-promoting activities and may also have initiating activities in carcinogenesis in the glandular stomach.

Tags: Male; Support, Non-U.S. Gov't

Descriptors: *Aldehydes--toxicity--TO; *Butanones--toxicity--TO; *Carcinogens; *Diacetyl--toxicity--TO; *Gastric Mucosa--drug effects--DE; *Glyoxal--toxicity--TO; *Stomach Neoplasms--chemically induced--CI; Animals ; DNA--biosynthesis--BI; Dose-Response Relationship, Drug; Enzyme Induction --drug effects--DE; Gastric Mucosa--metabolism--ME; Ornithine Decarboxylase --biosynthesis--BI; Rats; Rats, Inbred F344; Time Factors

CAS Registry No.: 0 (Aldehydes); 0 (Butanones); 0 (Carcinogens); 107-22-2 (Glyoxal); 431-03-8 (Diacetyl); 9007-49-2 (DNA)

Enzyme No.: EC 4.1.1.17 (Ornithine Decarboxylase)

Record Date Created: 19851218

Record Date Completed: 19851218

?logoff hold

07jul04 09:20:15 User228206 Session D2194.2

$3.94 1.230 DialUnits File155

$15.12 72 Type(s) in Format 9

$15.12 72 Types

$19.06 Estimated cost File155

$0.50 TELNET

$19.56 Estimated cost this search

$19.56 Estimated total session cost 1.390 DialUnits

Status: Signed Off. (2 minutes)

09370220 PMID: 1629897

Motility as a factor in the colonisation of gnotobiotic piglets by Helicobacter pylori .

Eaton K A; Morgan D R; Krakowka S

Department of Veterinary Pathobiology, College of Veterinary Medicine, Ohio State University, Columbus 43210.

Journal of medical microbiology (ENGLAND) Aug 1992, 37 (2) p123-7, ISSN 0022-2615 Journal Code: 0224131

Contract/Grant No.: AI07938-02; AI; NIAID; DK39570-01A3; DK; NIDDK

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Non-motile variants of **Helicobacter pylori** (strain **26695**) occurred with a frequency of 1.6 (SD 0.4) x 10(-4) variants/cell/division cycle, and reversion to the motile form occurred with a frequency of less than 10(-7) variants/cell/division cycle. The two forms remained greater than 90% pure for up to 50 cell divisions and differed only in the presence or absence of motility and flagella. Bacteria were recovered from nine of 10 gnotobiotic piglets inoculated orally with motile H. **pylori** , but from only two of eight inoculated with the non-motile variant. The motile form survived for 21 days in infected piglets, but the non-motile variant survived for only 6 days. Bacteria recovered from piglets inoculated with the non-motile variant were non-motile. These data support the hypothesis that motility is a colonisation factor for H. **pylori** .

Tags: Human; Support, U.S. Gov't, P.H.S.

Descriptors: Flagella--physiology--PH; * **Helicobacter pylori** --pathogenicity--PY; Animals; Cell Movement; Colony Count, Microbial; **Electrophoresis** , Polyacrylamide Gel; Germ-Free Life; Swine; Variation (Genetics)

Record Date Created: 19920820

Record Date Completed: 19920820

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Designations:	26695 [KE26695]	**Isolation:**	stomach of patient with gastritis, United Kingdom [24488]
Depositors:	KA Eaton	**History:**	ATCC<<--KA Eaton <<--A.D. Pearson
Biosafety Level:	2	**Shipped:**	frozen
Growth Conditions:	ATCC medium: 18 Trypticase soy agar Alternate Medium: 260 Trypticase soy agar with defibrinated sheep blood Growth Conditions: microaerophilic Temperature: 37.0 C		
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References:	24478: Tomb JF, et al. The complete genome sequence of the gastric pathogen Helicobacter **pylori.** Nature 388: 539-547, 1997. PubMed: 9252185 24488: Schmitt MJ, Tipper DJ . K28, a unique double-stranded RNA killer virus of Saccharomyces cerevisiae. Mol. Cell. Biol. 10: 4807-4815, 1990. PubMed: 2201903

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09370220 PMID: 1629897

Motility as a factor in the colonisation of gnotobiotic piglets by Helicobacter pylori .

Eaton K A; Morgan D R; Krakowka S

Department of Veterinary Pathobiology, College of Veterinary Medicine, Ohio State University, Columbus 43210.

Journal of medical microbiology (ENGLAND) Aug 1992, 37 (2) p123-7, ISSN 0022-2615 Journal Code: 0224131

Contract/Grant No.: AI07938-02; AI; NIAID; DK39570-01A3; DK; NIDDK

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

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Subfile: INDEX MEDICUS

Non-motile variants of **Helicobacter pylori** (strain **26695**) occurred with a frequency of 1.6 (SD 0.4) x 10(-4) variants/cell/division cycle, and reversion to the motile form occurred with a frequency of less than 10(-7) variants/cell/division cycle. The two forms remained greater than 90% pure for up to 50 cell divisions and differed only in the presence or absence of motility and flagella. Bacteria were recovered from nine of 10 gnotobiotic piglets inoculated orally with motile H. **pylori** , but from only two of eight inoculated with the non-motile variant. The motile form survived for 21 days in infected piglets, but the non-motile variant survived for only 6 days. Bacteria recovered from piglets inoculated with the non-motile variant were non-motile. These data support the hypothesis that motility is a colonisation factor for H. **pylori** .

Tags: Human; Support, U.S. Gov't, P.H.S.

Descriptors: Flagella--physiology--PH; * **Helicobacter pylori** --pathogenicity--PY; Animals; Cell Movement; Colony Count, Microbial; **Electrophoresis** , Polyacrylamide Gel; Germ-Free Life; Swine; Variation (Genetics)

Record Date Created: 19920820

Record Date Completed: 19920820

humans.

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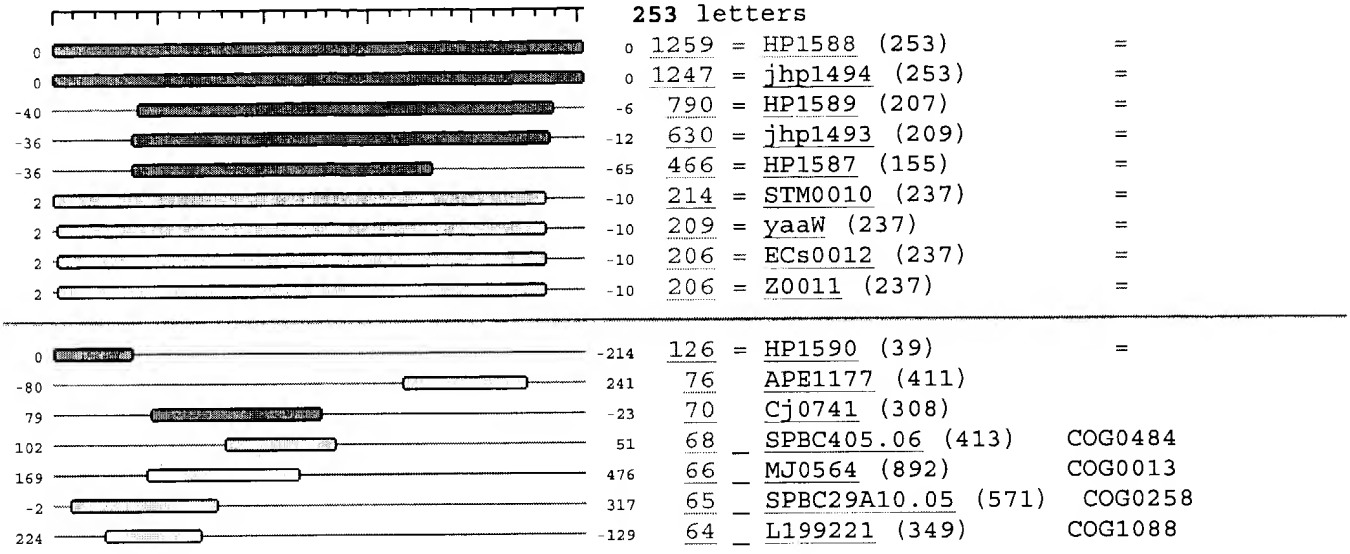
12 proteins	S	COG4735	Uncharacterized protein conserved in bacteria	Help

16 hits to 6 species



BLASTP 2.2.4 [Aug-26-2002]

Reference: Altschul, Stephen F., Thomas L. Madden, Alejandro A. Schaffer, Jinghui Zhang, Zheng Zhang, Webb Miller, and David J. Lipman (1997), "Gapped BLAST and PSI-BLAST: a new generation of protein database search programs", Nucleic Acids Res. 25:3389-3402.

Query= HP1588
(253 letters)

Database: myva
192,987 sequences; 59,019,183 total letters

Sequences producing significant alignments:	Score (bits)	E Value
HP1588	489	e-138
jhp1494	484	e-137
HP1589	308	6e-84
jhp1493	247	2e-65
HP1587	184	2e-46
STM0010	87	4e-17
yaaW	85	1e-16
ECs0012	84	3e-16
Z0011	84	3e-16

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HP1590                                                          53   6e-07
APE1177                                                         34   0.32
Cj0741                                                          32   1.9
SPBC405.06                                                      31   3.4
MJ0564                                                          30   4.9
SPBC29A10.05                                                    30   5.9
L199221                                                         29   8.6

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#           Length = 253
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#            MAYKYDRDLEFLKQLESSDLLDLFEVLVFGKDGEKRHNEKLTSSIEYKRHGDDYAKYAER
# Sbjct: 1   MAYKYDRDLEFLKQLESSDLLDLFEVLVFGKDGEKRHNEKLTSSIEYKRHGDDYAKYAER 60
#
# Query: 61  IAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYNKKTETTLIEQNMLSKILERS 120
#            IAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYNKKTETTLIEQNMLSKILERS
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#
# Query: 121 LEEMDDEEVKEMCDELSIKNTDNLNRQALSAATLTLFKMGGFKSYQLAVIVANAVAKTIL 180
#            LEEMDDEEVKEMCDELSIKNTDNLNRQALSAATLTLFKMGGFKSYQLAVIVANAVAKTIL
# Sbjct: 121 LEEMDDEEVKEMCDELSIKNTDNLNRQALSAATLTLFKMGGFKSYQLAVIVANAVAKTIL 180
#
# Query: 181 GRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLRLKTQQ 240
#            GRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLRLKTQQ
# Sbjct: 181 GRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLRLKTQQ 240
#
# Query: 241 ANGDKKSLQIESI 253
#            ANGDKKSLQIESI
# Sbjct: 241 ANGDKKSLQIESI 253
#
#
# >jhp1494
#           Length = 253
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#  Score =  484 bits (1247), Expect = e-137
#  Identities = 251/253 (99%), Positives = 252/253 (99%)
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# Query: 1   MAYKYDRDLEFLKQLESSDLLDLFEVLVFGKDGEKRHNEKLTSSIEYKRHGDDYAKYAER 60
#            MAYKYDRDLEFLKQLESSDLLDLFEVLVFGKDGEKRHNEKLTSSIEYKRHGDDYAKYAER
# Sbjct: 1   MAYKYDRDLEFLKQLESSDLLDLFEVLVFGKDGEKRHNEKLTSSIEYKRHGDDYAKYAER 60
#
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#            IAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYNKKTETTLIEQNMLSKILERS
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#
# Query: 121 LEEMDDEEVKEMCDELSIKNTDNLNRQALSAATLTLFKMGGFKSYQLAVIVANAVAKTIL 180
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#
# Query: 181 GRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLRLKTQQ 240
#            GRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLRLKTQQ
# Sbjct: 181 GRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLRLKTQQ 240
#
# Query: 241 ANGDKKSLQIESI 253
#            AN DKKSLQIES+
# Sbjct: 241 ANEDKKSLQIESV 253
```

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# >HP1589
#           Length = 207
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#  Score =  308 bits (790), Expect = 6e-84
#  Identities = 149/199 (74%), Positives = 170/199 (84%)
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# Query: 41  LTSSIEYKRHGDDYAKYAERIAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYN 100
#            +TSS EY+R+G DYAKY  RIAEELQ YG NSF +F + EGVLYKEILCD CD LKVNYN
# Sbjct: 1   MTSSTEYQRYGYDYAKYPRRIAEELQRYGGNSFMNFFRDEGVLYKEILCDACDHLKVNYN 60
#
# Query: 101 KKTETTLIEQNMLSKILERSLEEMDDEEVKEMCDELSIKNTDNLNRQALSAATLTLFKMG 160
#            KK++TTLIE+NMLS IL++SLE+M DEE++E+CDEL +KNT+ L +QALS A LTLF+MG
# Sbjct: 61  KKSDTTLIEENMLSSILQKSLEKMSDEEIRELCDELGVKNTNKLGKQALSTAALTLFRMG 120
#
# Query: 161 GFKSYQLAVIVANAVAKTILGRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPA 220
#            GFKSYQLA+IVANAV K I  RGLSL  N  LTR LS LTGP+GWIITGVWTAIDIAGPA
# Sbjct: 121 GFKSYQLALIVANAVIKAIFQRGLSLGANAALTRGLSILTGPIGWIITGVWTAIDIAGPA 180
#
# Query: 221 YRVTIPACIVVATLRLKTQ 239
#            YRVTIPACI+VATLRLK Q
# Sbjct: 181 YRVTIPACILVATLRLKAQ 199
#
#
# >jhp1493
#           Length = 209
#
#  Score =  247 bits (630), Expect = 2e-65
#  Identities = 122/204 (59%), Positives = 154/204 (74%), Gaps = 4/204 (1%)
#
# Query: 38  NEKLTSSIEYKRHGDDYAKYAERIAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKV 97
#            NE+LTS  EY+R+G DYAKY  RIAEELQ YG NSFA+F + EGVLYKEILCD CD L +
# Sbjct: 2   NEELTSLTEYQRYGHDYAKYPRRIAEELQRYGGNSFANFFRDEGVLYKEILCDACDHLDI 61
#
# Query: 98  NYNKKTETTLIEQNMLSKILERSLEEMDDEEVKEMCDELSIKNTDNL---NRQALSAATL 154
#            NYN+++ T+LIEQNMLSK+L+ SLE+M   E+KE+CD L + N D +   N+Q L A+ L
# Sbjct: 62  NYNERSATSLIEQNMLSKLLKDSLEKMSGREIKELCDGLGMPNIDKVIGENKQVLIASVL 121
#
# Query: 155 TLFKMGGFKSYQLAVIVANAVAKTILGRGL-SLAGNQVLTRTLSFLTGPVGWIITGVWTA 213
#            TLFK GG  SY LAV VA+A+ +  LG GL S+ G    L +TL  L GP+GW+ITG   +
# Sbjct: 122 TLFKAGGSHSYALAVAVADAMVRQTLGHGLSSVVGKVALKKTLDILAGPIGWVITGALVS 181
#
# Query: 214 IDIAGPAYRVTIPACIVVATLRLK 237
#            I++AGPAYRVT+PAC++VATLR K
# Sbjct: 182 INLAGPAYRVTVPACVLVATLRKK 205
#
#
# >HP1587
#           Length = 155
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#  Score =  184 bits (466), Expect = 2e-46
#  Identities = 93/147 (63%), Positives = 115/147 (77%), Gaps = 3/147 (2%)
#
# Query: 38  NEKLTSSIEYKRHGDDYAKYAERIAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKV 97
#            NE LT+S EYKR+G DYAKY  RIAEELQ+YG NSFA+F + EGVLYKEILCD CD LKV
# Sbjct: 2   NEDLTNSTEYKRYGHDYAKYPRRIAEELQHYGGNSFANFFRDEGVLYKEILCDACDHLKV 61
#
# Query: 98  NYNKKTETTLIEQNMLSKILERSLEEMDDEEVKEMCDELSIKNTDNL---NRQALSAATL 154
#            NYN+++ T+LIEQNMLSK+L+ SLE+M   E+KE+C+EL + N D +   N+Q L A+TL
```

```
# Sbjct: 62  NYNEESATSLIEQNMLSKLLKDSLEKMSRREIKELCNELGMTNIDKVIGENKQVLIASTL 121
#
# Query: 155 TLFKMGGFKSYQLAVIVANAVAKTILG 181
#            TLFK GG  SY LAV VA+A+ +  LG
# Sbjct: 122 TLFKAGGSHSYALAVSVADAMVRQTLG 148
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# >STM0010
#           Length = 237
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#  Score = 87.0 bits (214), Expect = 4e-17
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#            + Y +D DL+FL+       L D   +L   + G+ R +  L+ +  +K    H + + +
# Sbjct: 3   VTYLHDEDLDFLQHCSEEQLADFARLLTHNEKGKARLSSVLSHNELFKAMEGHPEQHRRN 62
#
# Query: 58  AERIAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYNKKTETTLIEQNMLSKIL 117
#             + IA E Q+YG +S A+ ++G G   Y+ IL DV  +LK+  +K   T  IEQ +L   L
# Sbjct: 63  WQLIAGEFQHYGGDSIANKLRGHGKQYRAILLDVAKRLKLKADKSMSTFEIEQQLLEHFL 122
#
# Query: 118 ERSLEEMDDEEVKEMCDELSIKNTDNLNRQALSAATLTLFK-MGGFKSYQLAVIVANAVA 176
#              + ++MD    +E    +  K ++      L     +L K +    S QL  I+    A
# Sbjct: 123 RHTWQKMDAAHKQEFLQAVDAKVSELEELLPLLMKDRSLAKGVSHLLSTQLTRILRTHAA 182
#
# Query: 177 KTILGRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLR 235
#             +I                  + L GPVG  + GV     ++G AYRVTIPA + +A LR
# Sbjct: 183 MSI---------LGHGLLRGAGLGGPVGAALNGVKA---MSGSAYRVTIPAVLQIACLR 229
#
#
# >yaaW
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#  Identities = 69/237 (29%), Positives = 112/237 (47%), Gaps = 16/237 (6%)
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#            Y  D DL+FL+       L +   +L   + G+ R +  L  +  +K    H + + +  +
# Sbjct: 5   YLNDSDLDFLQHCSEEQLANFARLLTHNEKGKTRLSSVLMRNELFKSMEGHPEQHRRNWQ 64
#
# Query: 60  RIAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYNKKTETTLIEQNMLSKILER 119
#             IA ELQ++G +S A+ ++G G LY+ IL DV  +LK+  +K+  T  IEQ +L + L
# Sbjct: 65  LIAGELQHFGGDSIANKLRGHGKLYRAILLDVSKRLKLKADKEMSTFEIEQQLLEQFLRN 124
#
# Query: 120 SLEEMDDEEVKEMCDELSIK-NTDNLNRQALSAATLTLFKMGGFKSYQLAVIVANAVAKT 178
#            + ++MD+E  +E    +  + N        L    L    +    S QL  I+    A +
# Sbjct: 125 TWKKMDEEHKQEFLHAVDARVNELEELLPLLMKDKLLAKGVSHLLSSQLTRILRTHAAMS 184
#
# Query: 179 ILGRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLR 235
#            +                  + L GPVG  + GV     ++G AYRVTIPA + +A LR
# Sbjct: 185 V---------LGHGLLRGAGLGGPVGAALNGVKA---VSGSAYRVTIPAVLQIACLR 229
#
#
# >ECs0012
#           Length = 237
#
#  Score = 84.0 bits (206), Expect = 3e-16
#  Identities = 68/237 (28%), Positives = 112/237 (46%), Gaps = 16/237 (6%)
#
# Query: 3   YKYDRDLEFLKQLESSDLLDLFEVLVFGKDGEKRHNEKLTSSIEYKR---HGDDYAKYAE 59
```

```
#            Y  D DL+FL+      L +   +L   + G+ R +  L  +  +K    H + + +  +
# Sbjct: 5   YLNDSDLDFLQHCSEEQLANFARLLTHNEKGKTRLSSVLMRNELFKSMEGHPEQHRRNWQ 64
#
# Query: 60  RIAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYNKKTETTLIEQNMLSKILER 119
#             IA ELQ++G +S A+ ++G G LY+ IL DV  +LK+  +K+  T  IEQ +L + L
# Sbjct: 65  LIAGELQHFGGDSIANKLRGHGKLYRAILLDVSKRLKLKADKEMSTFEIEQQLLEQFLRN 124
#
# Query: 120 SLEEMDDEEVKEMCDELSIK-NTDNLNRQALSAATLTLFKMGGFKSYQLAVIVANAVAKT 178
#            + ++MD+E  +E    +  + N        L    L    +    S QL  I+    A +
# Sbjct: 125 TWKKMDEEHKQEFLHAVDARVNELEELLPLLMKDKLLAKGVSHLLSSQLTRILRTHAAMS 184
#
# Query: 179 ILGRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLR 235
#            +                 + L GPVG  + GV     ++G +YRVTIPA + +A LR
# Sbjct: 185 V---------LGHGLLRGAGLGGPVGAALNGVKA---VSGSSYRVTIPAVLQIACLR 229
#
#
# >Z0011
#           Length = 237
#
#  Score = 84.0 bits (206), Expect = 3e-16
#  Identities = 68/237 (28%), Positives = 112/237 (46%), Gaps = 16/237 (6%)
#
# Query: 3   YKYDRDLEFLKQLESSDLLDLFEVLVFGKDGEKRHNEKLTSSIEYKR---HGDDYAKYAE 59
#            Y  D DL+FL+      L +   +L   + G+ R +  L  +  +K    H + + +  +
# Sbjct: 5   YLNDSDLDFLQHCSEEQLANFARLLTHNEKGKTRLSSVLMRNELFKSMEGHPEQHRRNWQ 64
#
# Query: 60  RIAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYNKKTETTLIEQNMLSKILER 119
#             IA ELQ++G +S A+ ++G G LY+ IL DV  +LK+  +K+  T  IEQ +L + L
# Sbjct: 65  LIAGELQHFGGDSIANKLRGHGKLYRAILLDVSKRLKLKADKEMSTFEIEQQLLEQFLRN 124
#
# Query: 120 SLEEMDDEEVKEMCDELSIK-NTDNLNRQALSAATLTLFKMGGFKSYQLAVIVANAVAKT 178
#            + ++MD+E  +E    +  + N        L    L    +    S QL  I+    A +
# Sbjct: 125 TWKKMDEEHKQEFLHAVDARVNELEELLPLLMKDKLLAKGVSHLLSSQLTRILRTHAAMS 184
#
# Query: 179 ILGRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTIPACIVVATLR 235
#            +                 + L GPVG  + GV     ++G +YRVTIPA + +A LR
# Sbjct: 185 V---------LGHGLLRGAGLGGPVGAALNGVKA---VSGSSYRVTIPAVLQIACLR 229
#
#
# >HP1590
#           Length = 39
#
#  Score = 53.1 bits (126), Expect = 6e-07
#  Identities = 26/39 (66%), Positives = 31/39 (78%)
#
# Query: 1   MAYKYDRDLEFLKQLESSDLLDLFEVLVFGKDGEKRHNE 39
#            MAY+YD DLEFLK+L SSDL DLF+ LV+ +DG  R NE
# Sbjct: 1   MAYRYDSDLEFLKRLSSSDLKDLFDALVYDEDGTLRMNE 39
#
#
# >APE1177
#           Length = 411
#
#  Score = 33.9 bits (76), Expect = 0.32
#  Identities = 20/61 (32%), Positives = 30/61 (48%), Gaps = 3/61 (4%)
#
# Query: 166 QLAVIVANAVAKTILGRGLSLAGNQVLTRTLSFLTGPVGWIITGVWTAIDIAGPAYRVTI 225
#            Q A I   ++AK  +G GL+  G + L   +  LT P+  +I G       + G A R+ I
# Sbjct: 86  QFASIALGSIAKLFVGVGLAAIGYKALAAVIGLLTMPLTIVIVGTTV---LLGLATRIGI 142
#
```

```
# Query: 226 P 226
#            P
# Sbjct: 143 P 143
#
#
# >Cj0741
#           Length = 308
#
#  Score = 31.6 bits (70), Expect = 1.9
#  Identities = 28/83 (33%), Positives = 41/83 (48%), Gaps = 3/83 (3%)
#
# Query: 47  YKRHGDDYAKYAE-RIAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYNKKTET 105
#            YK + + Y K+ + + A +    G   FASF K E   Y+E L  + DK   NY K  E+
# Sbjct: 126 YKNYTNLYEKWTQKKTAMDKIKTGEGYFASF-KEELQKYQEALAQL-DKENKNYEKIKES 183
#
# Query: 106 TLIEQNMLSKILERSLEEMDDEE 128
#             L+    L  I E+ LE+ +  E
# Sbjct: 184 GLVSDETLRAIYEKLLEQKEALE 206
#
#
# >SPBC405.06
#           Length = 413
#
#  Score = 30.8 bits (68), Expect = 3.4
#  Identities = 19/61 (31%), Positives = 29/61 (47%), Gaps = 7/61 (11%)
#
# Query: 82  VLYKEILCDVCDKLKVNYNKKTET-------TLIEQNMLSKILERSLEEMDDEEVKEMCD 134
#            V   +++CD C+   V++  K           T+ EQ MLS  + RS +E D    + M D
# Sbjct: 184 VTNSQVICDTCNGKGVSFRGKDRCKHCKGSGTVPEQRMLSFFVNRSAKENDKIIQRGMAD 243
#
# Query: 135 E 135
#            E
# Sbjct: 244 E 244
#
#
# >MJ0564
#           Length = 892
#
#  Score = 30.0 bits (66), Expect = 4.9
#  Identities = 21/74 (28%), Positives = 38/74 (50%), Gaps = 6/74 (8%)
#
# Query: 45  IEYKRHGDDYAKYAERIAEELQYYGSNSFASFIKGEGVLYKEILCDVCDKLKVNYNKKTE 104
#            ++Y++ GD+Y +   +I +    YG   F     GE  +Y  I  ++ +KLK +   K
# Sbjct: 214 MQYEKVGDNYKEIPLKIVD--TGYGIERFVWASTGEPTIYDAIFKNIVNKLKEDAGVKD- 270
#
# Query: 105 TTLIEQNMLSKILE 118
#                I++ +L+KI E
# Sbjct: 271 ---IDKEILAKITE 281
#
#
# >SPBC29A10.05
#           Length = 571
#
#  Score = 29.6 bits (65), Expect = 5.9
#  Identities = 24/74 (32%), Positives = 31/74 (41%), Gaps = 3/74 (4%)
#
# Query: 9  LEFLKQLESSDLLDLFEVLVFGKDGEK-RHNEKLTSSIE--YKRHGDDYAKYAERIAEEL 65
#           L  LK ++ S  ++ F     G DG    H    T + E  + +  D Y KYA   A  L
# Sbjct: 7  LGLLKPMQKSSHVEEFSGKTLGVDGYVWLHKAVFTCAHELAFNKETDKYLKYAIHQALML 66
#
```

```
# Query: 66 QYYGSNSFASFIKG 79
#           QYYG      F  G
# Sbjct: 67 QYYGVKPLIVFDGG 80
#
#
# >L199221
#           Length = 349
#
#  Score = 29.3 bits (64), Expect = 8.6
#  Identities = 16/49 (32%), Positives = 24/49 (48%), Gaps = 1/49 (2%)
#
# Query: 25  EVLVFGKDGEKRHNEKLTSSI-EYKRHGDDYAKYAERIAEELQYYGSNS 72
#            E  + G DGEK + E L   +    +  DDY +  +R   +L+Y   NS
# Sbjct: 249 ETYLIGADGEKNNKEVLEDILTRMGKDKDDYDRVTDRAGHDLRYAIDNS 297
#
#
  Database: myva
    Posted date:  Sep 16, 2002  2:25 PM
  Number of letters in database: 59,019,183
  Number of sequences in database:  192,987

Lambda     K      H
   0.317    0.134    0.373

Lambda     K      H
   0.267   0.0410    0.140


Matrix: BLOSUM62
Gap Penalties: Existence: 11, Extension: 1
Number of Hits to DB: 26,931,837
Number of Sequences: 192987
Number of extensions: 1076147
Number of successful extensions: 4003
Number of sequences better than 10.0: 45
Number of HSP's better than 10.0 without gapping: 15
Number of HSP's successfully gapped in prelim test: 30
Number of HSP's that attempted gapping in prelim test: 3974
Number of HSP's gapped (non-prelim): 48
length of query: 253
length of database: 59,019,183
effective HSP length: 107
effective length of query: 146
effective length of database: 38,369,574
effective search space: 5601957804
effective search space used: 5601957804
T: 11
A: 40
X1: 16 ( 7.3 bits)
X2: 38 (14.6 bits)
X3: 64 (24.7 bits)
S1: 41 (21.6 bits)
S2: 64 (29.3 bits)
S2: 64 (29.3 bits)
```